158

BRAIN AND INTELLIGENCE

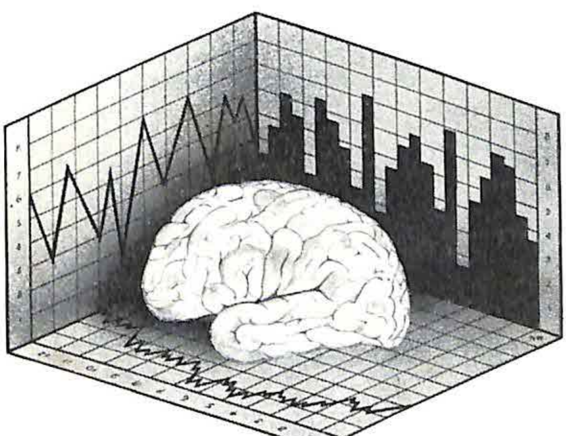
FIG. 1.—First recorded case history of frontal-lobe injury. From the Edwin Smith Surgical Papyrus, about 1700 B.C. (Taken from Breasted.) For a translation and discussion of the case history by the late Professor Henry Breasted see page 103 of this monograph.

BRAIN AND INTELLIGENCE

A Quantitative Study of the Frontal Lobes

BY

WARD C. HALSTEAD



THE UNIVERSITY OF CHICAGO PRESS · CHICAGO

THE UNIVERSITY OF CHICAGO PRESS, CHICAGO 37
Cambridge University Press, London, N.W. 1, England
The University of Toronto Press, Toronto 5, Canada

Published 1947. Third Impression 1956. Composed and printed by
THE UNIVERSITY OF CHICAGO PRESS, *Chicago, Illinois, U.S.A.*

PREFACE

When one views the progress of biology in retrospect, the broad truth stands out that there has been a continuity of development in biological thought and interpretation. The new proceeds out of the old, but is genetically related to it.—William Locy, 1908.

For all our accumulated knowledge concerning the details of brain structure the universals of mental function are, for the most part, lacking. Thus psychology is today the least abstract of the sciences, yet it is doubtless the most abstruse. Psychology remains concrete or particularistic in its implementable insights because nowhere has it yet been proved possible to distinguish clearly between those psychophysiological events which are means or process and those which are ends. For the student of behavior the general field of medicine offers a proving ground par excellence on which psychological hypotheses can be brought to terms with reality. As man undergoes dissolution of his functions under stress of time or of disease, so is he constructed. Illness of mind and body is compatible with neither "empty organisms" nor *ad hoc* philosophies. This is not to suggest that medicine has materially advanced our understanding of mental functions. In his impressionistic approach to behavior the clinician of today enjoys but slight advantage over Galen.

There are more than enough brain-injured people in the modern world to permit resolution of every fundamental question concerning the human mind, could this material but be brought under adequate study. Our inertia in this regard need scarcely be attributed to any lack of altruism on the part of brain-injured individuals or to the absence of an ethical framework within which such studies might be pursued. Our professionalized idols of the cave have in the past amply guaranteed our continued ignorance.

This monograph is addressed to those investigators and students in medical and nonmedical sciences whose problems transcend professional boundaries. Neurologists, psychiatrists, psychologists, and psychoanalysts will find here material relevant to their central concepts concerning man as organism. It is hoped that they, as well as educators and general biologists, will read the work as a whole before passing judgment.

The studies reported herein were begun in 1935 under the auspices of the Otho S. A. Sprague Memorial Institute and of the Division of Psychiatry, Department of Medicine, the University of Chicago, during the writer's tenure of a National Research Council Fellowship in psychology. In the subsequent twelve years of full-time research a substantial body of evidence has been accumulated concerning the behavioral effects of brain lesions in man.

While progress reports have been published in various journals from time to time, this monograph attempts to interpret and evaluate a major section of the findings. The quantitative results are based upon individual examinations of approximately two hundred and fifty subjects: neurosurgical patients, normal control individuals, normal individuals under experimental stresses, neuropsychiatric patients, and individuals with closed-head (brain) injuries. A battery of twenty-seven behavioral indicators was employed in the general investigation. Administering and scoring this battery averaged about fifteen hours per individual subject.

The investigation was carried out within a general hospital associated with a medical school. The members of this, the only full-time medical faculty in the United States, were consulted freely throughout to obviate errors in the basic data and in the interpretations. Drs. Percival Bailey, Paul C. Bucy, A. Earl Walker, and Richard B. Richter referred most of the neurological and neurosurgical patients. Drs. Emmet Bay and Wright Adams in cardiology, Dr. Allan Kenyon in endocrinology, and Drs. Hugh T. Carmichael and Henry W. Brosin in psychiatry similarly supported our efforts by referring patients for study. Referrals from other physicians are noted in the text.

Several behavioral indicators were developed and employed in these studies for the first time. Each of these bears the writer's name in the manuscript. It seemed desirable to risk the charge of professional immodesty in the interest of establishing a limited liability, i.e., scientific responsibility for prescribed sets of test conditions. The detailed specifications for each of these indicators will be made available to responsible workers in the field. All original data, brain sections (including serial sections through both hemispheres of Case 1, alternately stained for cells and fibers), correlations, and factor analyses are available for examination in this laboratory.

The Committee on Publications in Biology and Medicine as well as the technical divisions of the University of Chicago Press have contributed much to the shaping of this monograph into its present form.

Personal acknowledgments would necessarily be incomplete were space unlimited. But as teachers, friends, and colleagues, the writer's associations with Professors Franklin S. Fearing, Heinrich Klüver, C. Judson Herrick, and Karl S. Lashley have from the beginning made the present quest seem worth while.

Professors L. L. Thurstone and Karl Holzinger have read the statistical portions of the manuscript and have independently prepared the factor analyses of our data.

It is a pleasure to acknowledge here the substantial assistance of my research associates and laboratory assistants. These have included Dr. Paul Settlage, George Knox, George Sacher, Thomas Brill, Persa Bell, Ralph Reitan, James Roach, Matthew Hughes, Carolyn Ruhland Carmichael, Norma Tryner, Janet Schneider, Fay Trolander Brill, Evelyn Sherwood Whitehorn, Joyce Goodfellow White, Angela Peyraud Hinton, Dorothy Murphy Ford, and Margaret Andel Stevens.

WARD C. HALSTEAD

September 25, 1947

TABLE OF CONTENTS

LIST OF ILLUSTRATIONS

LIST OF TABLES

PART I

THE STRUCTURE OF BIOLOGICAL INTELLIGENCE

CHAPTER I

A MAN ON HORSEBACK

THE story to be told in the following pages concerns a twelve-year search in a psychological laboratory for "a man on horseback." The reader who may have observed over the years—sometimes with polite skepticism—the flowering of laboratory insight into those distant and not so distant cousins of man, the white rat, guinea pig, cat, dog, monkey, and the higher apes, will not be surprised that psychologists are now taking on "horses" and even "horses with riders." But the present work is only incidentally a study in comparative psychology. The allusion is to Freud's picturesque simile concerning basic structures in personality. Nearly a quarter of a century ago, he wrote: "The functional importance of the ego is manifested in the fact that normally control over the approaches to motility devolves upon it. Thus in its relation to the id it is like a man on horseback, who has to hold in check the superior strength of the horse; with this difference, that the rider seeks to do so with his own strength while the ego uses borrowed forces" (157).[1]

What is the basic plan or structure of the ego, and whence come its forces? Unfortunately for biological science, Freud left but a fragmentary outline as answers to these important questions. But there can be little doubt that he conceived of the ego as having definite form or structure. From his analysis of dream processes he not only projected a skeletal form for the ego, he entertained a notion for it of "psychic locality," a concept more familiarly known today as "localization of function" (158). It is regrettable that Freud's research material was not suitable for more extended excursions into the problem of localization of function in the brain. But developments in modern brain surgery in a sense came too late. Thus, it has been left to others to provide a neuropsychological description of the ego and to elucidate its mechanisms.

It may be noted in passing that the twentieth century has not elsewhere produced implemented insights into ego functions. The charge that this is an age of "anti-intellectualism" has not yet been successfully challenged. In psychiatry, the formalistic attempts of Kraepelin (108), the dynamics

[1] Numbers in parentheses indicate references cited in References and Bibliography at end of book.

of Bleuler (43), Janet (246), Rorschach (349), and Goldstein (176), for example, have produced no solid basis for a biological view of the human mind. In further testimony of this fact comes the recently published hundredth-anniversary volume of the American Psychiatric Association: *One Hundred Years of American Psychiatry* (186). Intended as "a historical synthesis of a century of American psychiatric evolution," this work is notable in that, while chapters are provided on the contributions of psychology and on the influence of psychiatry on anthropology, no chapter appears honoring the contributions of neurology by attempting to relate normal and pathological mind to the brain.

It appears that Freud's successors in the psychoanalytic idiom have made more progress in elucidating the "horse" than in advancing our understanding of the "rider." This disproportion becomes conspicuous when we read that "every neurosis and every psychosis represents *a failure of the ego in performing its function of securing adequate gratification for subjective needs under the existing external conditions.* This statement is valid for all forms of mental disturbance—whether they are caused by demonstrable organic damage to the brain tissue due to organic, chemical, bacterial causes or are psychogenic disturbances which develop as a result of traumatic life experiences. Psychotherapy in the latter type of cases aims to restore this ability to the ego by psychological means" (Alexander and French [6, p. viii]) and then find the ego characterized in two brief paragraphs—in somewhat less than analytic detail—as consisting of a perceptive faculty, an executive faculty, and an integrative function (6, p. ix). In view of such global specification of the ego, the prime target of psychotherapy, it perhaps is not surprising to find that the effective mechanisms in psychotherapy have remained obscure: "Like most psychoanalysts, we have been puzzled by the unpredictability of therapeutic results, by the baffling discrepancy between the length and intensity of a treatment and the degree of therapeutic success" (6, p. v).

Nor have academic psychologists fared better. Experimentalists have, on the whole, ignored the challenge of psychopathology, preferring the scientific respectability of the "well-controlled," if usually unvalidated, experiment. The mental-testing movement, on the other hand, largely uncontaminated in the beginning by "brass instruments," has become more and more dependent in recent years upon the calculating machine. From such investigators as Ebbinghaus (118), Binet (38), Terman (393), Stern (383), Burt (71), Stoddard (386), Tredgold (406), Thorndike (397), Spearman (378), and Thurstone (401), for example, have emerged the concept of the "intelligence quotient" (I.Q.) and "factor analysis," an investiga-

tive tool. The biological significance of each of these contributions remains largely undemonstrated, although the current sociological ramifications of both are numerous. As an index for scaling human mentality, the I.Q. has been widely used in the classroom and in the psychiatric clinic. In both, and especially in the latter, it is commonplace to observe marked disparities between the measured intelligence of an individual patient and his apparent usable intelligence. The former may be very high and the latter low or vice versa. This discrepancy becomes a patent absurdity in the case of brain-injured individuals. Evidence is now on record to the effect that surgical removal of one or both prefrontal lobes—that is, a mass of brain substance constituting about one-fourth of the total cerebrum—may not significantly alter the I.Q. (184, 209, 202).

But what of the neurologists, who must have approached the general problem "with their feet on the ground"? Here our summary of the situation can be brief indeed. The impressionistic study of lower animals with experimental brain lesions by such investigators as Flourens (143), Munk (328), Goltz (182), Hitzig (232), Flechsig (137), Loeb (296), Broca (55), Von Monakow (318), and Bianchi (36) has likewise yielded only confusion. As we shall see in a later chapter, this confusion has persisted in the face of quantitative behavioral methodologies introduced by comparative psychologists such as Franz (150), Lashley (286), and Klüver (271).

In general, psychologists have long despaired of creating a satisfactory definition of intelligence (396). Since, in one sense, "nothing follows from a definition," this lack presents no handicap to a scientific attack upon the problem. Nor has it stood in the way of hundreds of separate investigations. Some years ago Thorndike proposed that intelligence be conceived of as "that which intelligence tests measure" (396). This circular, if nonetheless "operational," definition of intelligence has served to keep the doors open to science and has permitted the individual investigator great freedom in exploring the relevant phenomena. Further, it has perhaps stimulated industry and ingenuity in compounding mental tests. The term "psychometric intelligence" is convenient and in line with Thorndike's definition for establishing a distinction between the biological and the sociological approach to the nature of intelligence. It should be noted that this distinction is a methodological one. Its acceptance for purposes of analysis of the problem need not commit the reader ultimately to any such dichotomy.

Abilities versus functions.—Although questions concerning animal and human intelligence were widely debated by biologists during the seventeenth, eighteenth, and nineteenth centuries (47), the intelligence test as

an instrument for scaling human mentality appeared in response to a practical sociological problem. In 1904 the minister of public instruction appointed Alfred Binet, director of the Laboratory of Physiological Psychology in the Sorbonne, as a member of a commission on special classes in the schools of Paris. A survey revealed that many children were failing in the school work for their age. Was failure in each case due to inadequate effort, to mental deficiency, or to other causes? Binet set out to find an answer. With Simon, he began devising simple objective tests which, when pooled into a battery, came to be known as the Binet-Simon Scale for Intelligence (38). "What mental abilities bearing upon performance in school does the normal child possess at different ages in which the mentally defective child may be deficient?" is the question to which Binet addressed his efforts. It should be noted that by casting his research problem in this form Binet provided himself with a specific, sociological criterion against which to validate his measuring device for mental abilities. His mental tests could be said to be valid if they successfully delineated among children the ability to perform a certain task: school work for their age. Likewise, his scale could be said to be reliable if repeated measurement of any child of given age yielded essentially the same result. Binet adopted a statistical approach and made use of those statistical techniques which were available to him at the time in the solution of his problem. He examined the reactions to specific test stimuli of hundreds of children through the age range of three to twelve years. With conformity in school work as a reference point, he was able statistically to locate the average child of normal "intelligence" for its age. An empirical basis was thus provided for selecting and arranging the subtests of the scale for increasing age levels of the average child. It is a matter of historical interest that Binet's scale, thus constructed, seemed to work. By applying it to scholastically retarded children, he found many instances of mental retardation as an underlying cause. Binet's scale, as modified by Terman (the Stanford-Binet Test of Intelligence), has become the most adequately standardized and the most widely employed individual test for intelligence in current use.

Except by way of speculation, Binet nowhere raised the question of the biological nature of intelligence. Nor, in general, have his successors in the field of human mentality. Among these, the factor analysts follow Binet in choosing scales which differentiate statistically the ability to perform certain tasks under certain conditions (e.g., in education or industry). The scales are accepted as valid if they work appreciably better than chance.

It is not yet generally recognized that even when this practical, statistical goal is achieved, biological insight into the functions involved does not magically appear. Some proponents of factor analysis have noted this hiatus. Guilford (185), for example, has written: "It may have to be admitted that the same factor does not carry the same relative weight for a task in different individuals or in the same individual at different times. . . . . It may even be true that the same test does not draw upon the same abilities when it is relatively easy for a population and when it is relatively difficult. [Godfrey] Thomson held similar views when he pointed out that the same task did not always call upon the same set of factors in the same individual at different times, or in different individuals at the same time" (p. 469). But, thus far, the factor analysts have made no systematic attempt to validate their scales in terms of basic biological functions of the organism. Like Benjamin Franklin, who is said to have cut a large hole in the barn door for big cats to pass through and a small hole for kittens, they point their scales at first one criterion and then another without concern for fundamental biological insight.

Biological intelligence.—Is there a biology of intelligence which is characteristic of the normal human nervous system wherever it is found? Does it contribute to man's survival as an organism? Is it different in degree, in kind, or in both from that possessed by members of other surviving species? Is it unitary or comprised of multiple factors? More practically, can convenient indices be found which, like blood pressure, accurately reflect the normal and pathological range of variance for the individual? Is there a pathology of biological intelligence which is of significance to psychiatry and to our understanding of normal behavior?

These and many other questions follow directly from an affirmative answer to the question of the existence of biological intelligence. On the other hand, a negative answer would render virtually hopeless any rational solution to many of these problems, including the problem of localization of mental functions in the brain. As a limiting case, the degrees of freedom might coincide with the units of population. Such a situation would permit of as many solutions as there are individuals in the universe, resulting in scientific indeterminacy.

From the standpoint of parsimony, the affirmative hypothesis offers a rational point of departure and has been adopted in the present studies. Thus it is that the first section of this monograph is devoted to the structure of biological intelligence, while the second section is concerned with evidence for localization of its functions in the brain.

CHAPTER II

PSYCHOMETRIC INTELLIGENCE

ALL theories of intelligence are in a sense psychological, but it will aid our later understanding if we survey the various conceptions of the nature of intelligence that have been seriously set forth by professional psychologists. By thus limiting our inquiry for technical reasons, we are excluding at the outset the contributions to the subject of many of the ablest thinkers of the past: men whose insight and understanding is recorded as the cultural heritage of the civilized world. A scientific inquiry into the nature of intelligence has as its goal that of science in general. Such inquiry is nonetheless necessary, if we are to find implementable truths, even though its results but affirm that which men of wisdom have known all along.

Those theories which seek a naturalistic interpretation of intelligence may be grouped roughly into four classes. While such classification is to a considerable extent arbitrary, it may, nevertheless, help in examining the neurological implications of particular theories.

1. *The unit-factor theory* conceives of intelligence as a single and separate factor or attribute of mind. It pervades specific abilities to a greater or less extent but is distinct from them. It is commonly referred to as "general intelligence," thus further emphasizing its unitary status. The evidence most commonly cited as proof of its existence is that specific abilities may be absent in the presence of high general intelligence or present in the absence of high intelligence. The unitary conception of intelligence has gained support from a variety of sources, and only in recent years has it been seriously challenged by alternate views. The unitary conception was enunciated in somewhat different terms by Ebbinghaus (118) in Germany and Binet (38) in France near the beginning of the present century. Both men proposed specific suggestions for the measurement of intelligence which, in modified form, are currently employed to some extent.

Ebbinghaus (118) reported the results of his extensive impressionistic analysis of those performances in ordinary life which seem most likely to involve the use of intelligence. He concluded that the critical ability required by intelligence was the ability to combine or integrate independent impressions into unitary or meaningful wholes. The essential nature of

intelligence was, thus, combination. This capacity, Ebbinghaus believed, could be rendered defective in a child through injuries at birth or through disease.

Binet (38), seeking an answer to retardation of school children, supplemented impressionistic analysis of mental behavior with simple experiments. He concluded that the essential characteristic of intelligence, of the greatest importance in practical life, is judgment. A person lacking in judgment may be feeble-minded or an imbecile, while a person with good judgment is never such. Not only is judgment a separate unity, it also governs the use of the special abilities.

Terman (393), who has modified the Binet scales into the well-known Stanford-Binet test, conceives of the unitary capacity as the capacity for abstract thinking. He provides no analyses of thinking processes which identify this form of behavior as the essence of intelligence. He likewise fails to specify criteria of abstract thinking by which to detect the pathologies of such thinking characteristic of large segments of the populations of our mental hospitals.

Stern (383), who first suggested the use of the intelligence quotient, or I.Q., as an index of mental development (i.e., mental age divided by chronological age), defines intelligence as "adaptability to new situations." He offers no clues, however, which enable us to distinguish pathologies of adaptability represented, for example, by the neuroses.

Franz and Gordon (151) emphasize the naturalistic character of the unitary capacity. They wrote in 1933: "Intelligence is *innate*, since it is inborn and inheritable, and, as such, is the capacity to learn, rather than learning. Further, intelligence is *general*, rather than specialized, and, hence, is to be distinguished from special abilities. Finally, intelligence is *intellectual capacity*, and, therefore, it is to be differentiated from traits of temperament or character that are primarily on an emotional basis" (p. 40).

A similar view was expressed by Burt *et al.* (71) in 1934: "By intelligence, the psychologist understands *inborn, all-round intellectual ability*. It is inherited or at least innate, not due to teaching or training, it is intellectual, not emotional or moral, and remains uninfluenced by industry or zeal; it is general, not specific, that is, it is not limited to any particular kind of work, but enters into all we do or say or think" (pp. 28–29).

Stoddard (386), who denies that intelligence is totally independent of learning and experience, offers a somewhat more sociological definition: "Intelligence is the ability to undertake activities that are characterized by (1) difficulty, (2) complexity, (3) abstractness, (4) economy, (5)

adaptiveness to a goal, (6) social value, and (7) the emergence of originals, and to maintain such activities under conditions that demand a concentration of energy and a resistance to emotional forces" (p. 4). An obvious difficulty presented by Stoddard's definition lies in the fact that the characteristics which he enumerates are themselves no more readily specifiable in psychological terms than intelligence itself. He thus exchanges one unknown for at least seven or more.

The above examples are perhaps sufficient to indicate the great diversity of opinion which reigns among those psychologists who conceive of intelligence more or less as a unitary factor or primordial capacity of the organism. This view of intelligence is commonly shared by students of heredity, especially those concerned with the various pathologies of intelligence. Tredgold (406), for example, conceives of feeble-mindedness or "primary amentia" as a hereditary factor dependent upon germinal variations having their locus in the chromosomes. He defines amentia as follows: "A state of restricted potentiality for, or arrest of, cerebral development, in consequence of which the person affected is incapable at maturity of so adapting himself to his environment or to the requirements of the community as to maintain existence independently of supervision or external support" (pp. 8–9). It is now common medicolegal practice in this country to require that clinical impressions of feeble-mindedness be supported by low intelligence-test scores. Whatever the biological implications of the unit-factor theory may be, and these are by no means clear, the fact remains that a conception has developed of intelligence as a capacity of some kind which has a Gaussian distribution in the general population. Its valuation aspect is reflected by the now widely held notion that good intelligence is an important factor in social effectiveness and that low intelligence is an important factor in social ineffectiveness.

In the face of so many unknowns, Lashley (286), writing in 1929 from the point of view of biological psychology, was forced to conclude: "The concept of intelligence is becoming essentially a statistical one; it is the correlation between certain of the activities of the organism which are closely related among themselves and relatively independent of other activities. Among such groupings of activities, what correlations constitute intelligence, as opposed to other capacities? There is no accepted statement which really defines the concept" (pp. 11–12).

2. *The two-factor theory.*—If the concept of intelligence has become essentially a statistical one, much of the impetus for this direction has come from Spearman (375–80), who has pioneered the development of statistical tools for investigating the nature of intelligence. Basing his in-

terpretations upon the objective results obtained with mental tests when analyzed by his method of factor analysis, Spearman, in 1904, put forward the notion that all mental responses in the cognitive sphere are resolvable into two factors. One of these is common to every reaction and being thus general in nature is called G. The other is specific in nature and is called S. The S factors are independent of one another and are also independent of G. Spearman has described the S factors somewhat picturesquely as "specific mental engines" (378, p. 137). He conceived of G, on the other hand, "as the amount of a general mental energy" (378). Thus, G corresponds to a power or energy reservoir in the brain upon which the "specific mental engines" draw for their fuel. As a simple, deterministic, mechanistic scheme, no more forthright view of the biological nature of intelligence is to be found in the whole of the literature on the subject. Yet it is chiefly from the biological standpoint that the theory remains in the realm of speculation, for thus far no systematic program has appeared for testing the biological (neurological, biochemical, and neurophysiological) implications of this conception. Spearman cited no convincing evidence from these areas in support of his notion of G as "general mental energy." Nor did he consider the possibility that G is in actuality an attitudinal factor in the sense, for example, of Goldstein (175).

Until Spearman set forth his conception of G, the usual method of examining a table of intercorrelations, such as Table 3, page 40, had been by inspection. This impressionistic method of determining the trends of data was far too permissive to avoid controversies over interpretation. It was at this point that Spearman undertook the development of a rational mathematical solution of the problem which subsequently came to be known as "factor analysis." He observed that the correlations from mental-test data of various sorts tended toward a peculiar arrangement, when grouped by rank, which could be expressed in a definite mathematical formula. This formula is known as the "tetrad equation" and may be written as

$$r_{ap}\, r_{bq} - r_{aq}\, r_{bp} = 0,$$

where r stands for any correlation of the Pearson type and the subscripts a, b, p, and q indicate any four mental abilities or tests.

Spearman and his students have collected a considerable body of evidence from mental-test batteries, in which the tetrad equation has been satisfied and a general factor has thus been indicated. Notable exceptions have also appeared which have led in some instances to modifications of the original theory.

3. *The three-factor theory.*—One such modification has been proposed by Holzinger (235, 236), who, working with Spearman, found it necessary to postulate the existence of group factors in addition to G and S.

4. *Multiple-factor theory.*—Thurstone (398–403) has challenged the adequacy of the two-factor and three-factor theories on the grounds that they are too parsimonious to fit the evidence for multidimensionality of mind. He has proposed instead a general multiple-factor theory, which, he finds, includes the tetrad-equation situation as a special case. His method utilizes the fact that the correlation between two tests is the scalar product of the two test vectors. If the two tests are perfect, then their scalars are both unity so that the true correlation between the two tests is the cosine of the angular separation between the two vectors (398). He rotates the co-ordinate axes until he finds the smallest number of axes (factors) in terms of which the variance of the intercorrelations may be described.

While Thurstone's method has thus far been applied largely to mental-test data, there would seem to be no fundamental reason why it should not be applicable to multiple-factor problems which occur in several fields of biology and medicine. Thurstone has isolated by his method several "distinguishable cognitive functions" which he terms "primary mental abilities." Among these are a deductive factor (D), an inductive factor (I), a memory factor (M), a spatial factor (S), a number factor (N), a word factor (W), and a perceptual factor (P).

Independently of factor analysis, Thorndike (397) proposed a conception of intelligence which, in many respects, is analogous to the multiple-factor theory. Intelligence for him consists of a large number of interrelated or interconnected abilities which correspond to interconnections within the brain. The greater the number of such neural interconnections, the greater will be the intellectual (intelligence) level of the individual. Thorndike also proposed his well-known C.A.V.D. test, which he regarded as a valid measure for "some unified, coherent, fundamental fact in the world."

It is apparent that no generally accepted theoretical framework as to the nature of psychometric intelligence has thus far been developed in support of the many measuring devices which are now widely applied. There can be little doubt that the best of these devices can yield fairly reliable measures of something, but of what? Regardless of what X factor is involved, it is important to note that a relatively high degree or amount of X is compatible with such concomitants as superior social adjustment, inferior social adjustment, superior school work, inferior school work,

superior physical health, inferior physical health, well-adjusted personality, schizophrenia, good citizenship, criminality, distinguished intellectual achievement, absence of both prefrontal lobes, good judgment, relatively high-grade anoxia, maleness and femaleness, preschool age, and adulthood. Is X a single or a multiple factor? Is it predominantly environmentally determined, or is it a direct reflector of basic biological functions of the organism?

It is clear that for these and other important questions we cannot yet obtain a rigorous answer, although the tool of factor analysis supported by biological validation of the factors isolated looks promising for the future. But, for the present, the situation is fundamentally little different from that prevailing when Lashley (286) wrote in 1929: "The whole theory of learning and of intelligence is in confusion. We know at present nothing of the organic basis of these functions and little enough of either the variety or uniformities of their expression in behavior. The concepts are so poorly defined that it has not been possible even to imagine a program of physiological research which seemed likely to reveal more than superficial relationships" (p. ix).

CHAPTER III

CLINICAL INTELLIGENCE

PSYCHIATRISTS, psychoanalysts, neurologists, and psychologists have traditionally been concerned with the various aspects of adaptive behavior. It is of interest that only the psychologists have made systematic attempts to measure intelligence as a component of adaptive behavior. The neurologist early attempted to localize intelligence in the brain. But, as we shall see, from his efforts has failed to come fundamental agreement concerning the nature of intelligence. Both in the neurological clinic and in the laboratory study of experimental brain lesions in lower animals, the neurologist's working approach to intelligence has traditionally been impressionistic rather than mensurate.

The psychiatrist and the psychoanalyst, most directly concerned in our society with the care and treatment of pathologies of mind, have likewise been content largely with impressionistic evaluations of intelligence. Failure of these medical disciplines to develop a distinctive conception of intelligence along biological lines arises from various sources which need not be elaborated here. The historical fact remains, however, that, with direct access to patients who present every conceivable variation in intelligence, they have made no contribution to the measurement or elucidation of intelligence. Yet the need for measurement, at least, has been recognized. In recent years this need has been met by borrowing from the psychologist psychometric devices usually developed outside a clinical setting. It is not surprising that the result in many instances has been mismeasurement and misinterpretation of the pathologies of adaptive behavior. Certain fragmentary attempts to establish a theoretical framework for intelligence have been made under clinical auspices. For example, in the descriptive psychiatry of Kraepelin (108) the reverberations of mental illness throughout the higher mental processes were clearly recognized: "The material of experience, received through the different senses and clarified by attention, forms a basis for all further mental elaboration, and it is self-evident that both disturbances of apprehension and the inability to make a systematic choice in the impressions, must effect to a marked degree the character of all intellectual processes" (p. 23).

Kraepelin avoided the term "intelligence" but resolved the intellectual processes into an ascending hierarchy of functions. "All higher mental activity," he wrote, "depends largely upon memory" (p. 23). "The *formation of concepts* is the necessary condition for the fullest development of ideation. In normal life those elements of experience which are often repeated impress themselves more and more strongly, while the accidental variations of each individual experience are driven more and more into the background. The concepts thus developed are a sort of composite photograph or generalization of experience" (p. 29). "Judgment and inference are the most complex products of the intellect. Since perception, memory, the formation of concepts, and the association of ideas are their necessary preconditions, they will be more or less affected by every imperfection of these processes" (p. 47).

Bleuler (43), whose interpretative school of psychiatry in Burghölzli contributed much to the advancement of Freud's theories, early adopted what is in essence a multiple-factor theory of intelligence. He wrote: "Intelligence in any sense whatsoever is never a unit. There is no one who is eminent in all psychic fields, while most idiots naturally fail in all directions. Practical intelligence does not necessarily imply tenetical intelligence and vice versa" (p. 27). With Kraepelin (108), Freud (158), Janet (246), and others, Bleuler borrowed from England the mechanism of associationism as the vehicle for binding the diverse elements of intelligence together: "All these achievements (intellectual) are primarily dependent upon the *number of possible associations*. The greater the number of stones at our disposal, the greater the number of ideas and the finer the shades that we can express in the mosaic of our thinking" (41, pp. 23–24). The reader may find it of interest to compare the views of Thorndike (397) with those of Blueler.

Both Kraepelin and Bleuler emphasized the holistic character of mental illness—illness which involves the whole of personality, including the higher mental functions. For Kraepelin, the highest reaches of the intellect—judgment and reasoning—were never completely spared in mental illness because of their hierarchical position. For Bleuler, on the other hand, special forms of intelligence—"stones of the mosaic"—could be spared. It is of interest that Freud, who in founding psychoanalysis concerned himself with the study of the "total personality," agreed implicitly, at least, with Bleuler in this respect; for one frequently encounters in Freud's writings such phrases as "intelligent neurotic," "intelligent psychopath," etc.

Nowhere did Freud set forth a specific theory of intelligence per se. It

is clear in his discussion of the ego functions that intelligence is an important aspect of the "reality-testing" principle. But it is forever caught between the crossfire of the id forces and the superego forces. Its available energy waxes and wanes under dynamic stress. Freud thus sketched in barest outline a potentially biological view of intelligence. But such fragments do not constitute a theory of intelligence. Certain evolutionary trends in his thinking about intelligence are to be noted, however. Thus, toward the end, Freud (157) wrote in *The Future of an Illusion:* "We may insist as much as we like that the human intellect is weak in comparison with human instincts, and be right in doing so. But, nevertheless, there is something peculiar about this weakness. *The voice of the intellect is a soft one, but it does not rest until it has gained a hearing.*[1] Ultimately, after endlessly repeated rebuffs, it succeeds. This is one of the few points in which one may be optimistic about the future of mankind, but in itself it signifies not a little" (p. 93).

Goldstein (170–77), possibly in reaction against the mental-testing movement which he has deplored, has largely avoided the term "intelligence" in his writings. He has, however, suggested a view of behavior which contains—in much the same sense as does Freud's concept of the ego—many connotations if not denotations of intelligence. His views have developed over a period dating from his studies of brain-injured and psychiatric patients during and after World War I. His opinions have sometimes been misunderstood by those who would think of them as Goldstein's Laws rather than as conceptions of behavior having their origin in large part in clinical expediency. As Goldstein (175) states: "To the physician, the need for emphasis upon the practical is clearly evident. Even in his theoretical considerations, it is natural for him to be drawn in a practical direction because the problem of healing is the very heart of his activity" (p. 5). Goldstein postulates a dichotomous separation of behavior into the "concrete" and the "abstract." In this distinction he follows Ach (1), who had also contributed the concept of "determining tendency" as the predeterminer of level of adaptation. According to Goldstein, this predeterminer is attitudinal in character (175). He has recently described his dichotomy as follows:

> In "concrete" performances a reaction is determined directly by a stimulus, is awakened by all that the individual perceives. The individual's procedure is somewhat passive, as if it were not he who had the initiative. In "abstract" performances an action is not determined directly and immediately by a stimulus configuration but

[1] Italics mine.

by the account of the situation which the individual gives to himself. The performance is thus more a primary action than a mere reaction, and it is a totally different way of coming to terms with the outside world. The individual has to consider the situation from various aspects, pick out the aspect which is essential, and act in a way appropriate to the whole situation. True, this procedure may have various degrees of complexity. Sometimes the situation demands nothing more than a singling out of one property of an object, as, for instance, when we are asked to sort objects according to their colors. In the highest degree of complexity we have not only to apprehend objects by means of certain simple characteristics but to choose aspects for consideration in accordance with a certain task which demands a conceptual organization. *Even in its simplest form, however, abstraction is separate in principle from concrete behavior. There is no gradual transition from the one to the other.*[2] The assumption of an attitude toward the abstract is not more complex merely through the addition of a new factor of determination; it is a totally different activity of the organism. Perhaps it would be better not to designate both conditions by the term "behavior," since behavior connotes real activity and is especially well suited to the concrete performance. Abstraction represents, rather, a preparation for activity; it involves an attitude, i.e., an inner approach, which leads to activity. Therefore, it is better to speak of an attitude toward the abstract. Real action is never abstract; it is always concrete. The difference between the two conditions is shown in the difference between the processes which precede action. In the concrete situation, action is set going directly by the stimuli; in the situation involving the abstract, action is begun after preparation which has to do with consideration of the whole situation [pp. 59–61].

Goldstein's theory of "abstract" and "concrete" behavior presents unusual difficulties from the standpoint of scientific parsimony in methodology since "what is concrete for one individual can only be understood within the frame of reference for that particular patient, as it is related to his pre-morbid individuality and his changed capacity and the situation given" (175, p. 59). In his emphasis upon the ability to carry out "abstract thinking," Goldstein is close to the unit-theory of intelligence espoused by Terman (393). Although initially holistic in his neurological conceptions of "abstract" behavior, Goldstein has moved in recent years toward the regional localization theory and specifies the frontal lobes as the region of the brain most directly involved in such behavior (173).

The Swiss psychiatrist, Rorschach (349), while contributing no specific theory of intelligence per se, nevertheless did emphasize (as did Goldstein) the necessity of estimating intelligence within the setting of the total personality. His well-known ink-blot test constitutes the instrument by which he proposed to accomplish this task. The intelligence component of personality is to be recognized in the organization drive (*Assoziationsbetrieb*). The best reflector of this in the Rorschach protocol is the W (whole) responses of the individual. "The number of W is, before

[2] Italics mine.

all, index to the energy at one's disposal for the organization drive" (p. 63). Closely related to the W response in intellectual significance is the M (*Bewegung*) response.

Rorschach believed that the well-integrated personality is reflected by an orderly sequence in the protocol. For individuals of good intelligence this succession is of the order of 8W, 23D, 3Dd for a protocol of thirty-four responses (cf. 30 and 349).

Whereas professional psychologists have dealt with intelligence as a distinct, scalable attribute of mind, psychiatrists and psychoanalysts tend to conceive of intelligence as embedded in personality. The dynamics of intelligence, according to this latter view, can be estimated only as a part of the appraisal of the total personality. This view arises less from specific analysis of the nature of intelligence than from observations of pathological forms of behavior.

CHAPTER IV

NEUROLOGICAL CONCEPTIONS OF INTELLIGENCE

NEUROLOGICAL conceptions of intelligence have for the most part developed along impressionistic lines with little attempt at measurement. They have been influenced more or less directly by mechanistic conceptions of the "reflex arc" elaborated during the seventeenth, eighteenth, and nineteenth centuries, by the "holistic" physiology of the central nervous system developed by the distinguished French physiologist, Marie Jean Pierre Flourens (1794–1867), and by the pseudo-science of phrenology initiated by the French anatomist, Franz Joseph Gall (1758–1828). For the historical background of these influences in biology the reader is referred to Fearing (125), Fulton (160), and Boring (47) and to the original works cited in the following discussion.

It is possible to classify the neurological theories of intelligence into three fairly distinct points of view.

1. *The holistic theory.*—Flourens (143) in 1842 set forth the doctrine that intelligence is an inseparable function of the activity of the entire cerebrum:

> En effet, non seulement toutes les perceptions, toutes les volitions, toutes les facultés intellectuelles résident exclusivement dans cet organe [brain], mais toutes ces facultés y occupent la même place. Dès qu'une d'elles disparaît par la lésion d'un point donné du cerveau proprement dit, toutes disparaissent; dès qu'une revient par la guérison de ce point, toutes reviennent. La faculté de percevoir et de vouloir ne constitue donc qu'une faculté essentiellement une; et cette faculté une réside essentiellement dans un seul organe [p. 244].

Because of the widespread current acceptance of Gall's doctrines of phrenology, Flourens' view precipitated much bitter controversy, most of which added nothing to the clarification of basic problems. It did, however, attract the talents of various experimentalists.

Goltz (182) studied the effect of experimental lesions in the brains of dogs. He agreed with Flourens that intelligence cannot be parcellated into separately localized functions but is rather a function of the brain as a whole. Sensory deficits produced by localized lesions produce distinctive alterations in behavior which, however, are not to be confused with alterations of intelligence. Much in the sense that Flourens anticipated the views of Spearman, Goltz anticipated some of the conceptions currently

held by K. S. Lashley, whose contributions are considered elsewhere in this chapter. He seems to have been the first to note in a series of experimental animals a direct relation between the extent of the brain lesion and the resulting dementia. He wrote:

> Wir konnten von Neuem bestätigen, dass jede erhebliche Verletzung der Rinde beider Grosshirnhälften die Intelligenz des Thieres in hohem Masse Schädigt, Blödsinn zur Folge hat. Flourens' Satz, dass nach theilweiser Wegnahme des Grosshirns die Intelligenz nicht herabgedrückt werde, ist nur theilweise richtig. Nach Entfernung der Rinde einer Grosshirnhälfte ist allerdings die Intelligenz in manchen Fällen nicht merklich beeinträchtigt. So wie aber beide Halbkugeln in einiger Ausdehnung verstümmelt sind, ist die Störung der Intelligenz stet deutlich und dauernd vorhanden. Im Allgemeinen kann man wohl behaupten, dass der Grad des Blödsinns nach Verstümmelung der Grosshirnrinde gleichen Schritt, hält mit der räumlichen Ausdehnung der Verletzung [p. 33].

In support of his notion of a quantitative relationship between dementia and size of lesion, Goltz pointed out that removal of the posterior quadrants of the brain, in keeping with their larger areas, produced a degree of dementia somewhat greater than that produced by removal of the anterior quadrants. Removal of three quadrants resulted in even greater dementia, and maximal dementia was seen after removal of four quadrants. Goltz interpreted the fundamental defect in his animals as a defect in attention. Following a cerebral lesion, the animal is unable to attend properly to groups of stimuli and thus fails to achieve integration or intelligent behavior. This would seem to imply that intelligence is dependent upon some function which is common to all parts of the cerebrum and which varies only in quantity. In this sense the view is perhaps most compatible with the unit theory of intelligence discussed in a preceding section.

Ferrier (127), working with dogs and monkeys, reached conclusions somewhat similar to those of Flourens and Goltz. He wrote:

> We have, therefore, many grounds for believing that the frontal lobes, the cortical centres for the head and ocular movements, with their associated sensory centres, form the substrata of those psychical processes which lie at the foundation of the higher intellectual operations. It would, however, be absurd to speak of a special seat of intelligence or intellect in the brain. Intelligence and will have no local habitation distinct from the sensory and motor substrata of the cortex generally. There are centres for special forms of sensation and ideation, and centres for special motor activities and acquisitions, in response to and in association with the activity of sensory centres; and these in their respective cohesions, actions, and interactions form the substrata of mental operations in all their aspects and all their range.
>
> We have not yet found, nor are we likely to discover, any simple formula to express the relation between brain and mind. It is not a mere matter of brute weight or quantity, absolute or relative; though there is no doubt that in animals of the same order a brain below a certain standard of weight is incompatible with normal intelligence [p. 467].

Loeb (296) not only carried the holistic view of intelligence into the present century but in some respects anticipated neurophysiological developments which were to come several decades later. Working primarily with dogs, he reached the conclusion that intelligence is a function of the cerebrum as a whole. He summarized his position as follows:

> If we wish to produce psychic disturbances by a lesion of the brain, we must destroy extensive parts of *both* hemispheres. Operations in one hemisphere alone, and even the destruction of an entire hemisphere, have no such effect.
>
> It has been claimed that the intellect is the function of special parts of the brain. Hitzig and others assumed that the frontal lobes of the cerebral hemispheres are the organs of attention. I have repeatedly removed both frontal lobes in dogs. It was impossible to notice the slightest difference in the mental functions of the dog. There is perhaps no operation which is so harmless for a dog as the removal of the frontal lobes [p. 274].

Loeb suggested that cerebral integration is brought about through the linkage of functional periodicities among the parts of the cortex through resonance. Intelligence is thus dependent upon a quantitative property of the cerebrum, namely, its capacity for resonance, a view which is compatible with the unit theory of intelligence. It should be noted that Loeb, like Goltz and Ferrier, avoided the complication of specific abilities in his conception of intelligence by relegating them to the level of simple sensory-motor arcs.

Feuchtwanger (128) examined four hundred cases of gunshot injury of the brain from World War I. His cases were divided equally between lesions within and outside the frontal lobes. He employed methods which were to some extent psychological in his analysis of the behavioral deficits. While he nowhere attempted a rigorous definition of intellect, he nevertheless reached the conclusion that injury to the frontal lobes per se does not produce a disturbance in intellect more frequently than does injury occurring to portions of the cerebrum outside the frontal lobes. Thus Feuchtwanger's clinical cases seemed to support the interpretations of Goltz, Ferrier, Munk, and Loeb, or the unit theory of intelligence. It is extremely doubtful, however, that Feuchtwanger's results can be taken to support any generalization concerning brain function. Personality disturbances, observed to be widespread in his group of chronically hospitalized, brain-injured soldiers, were doubtless reinforced by the flagging morale of a surrendered army. Thus, important differences pointing to localization may have been obscured in his results.

While certain improvements in operative technique had occurred since the days of Flourens, impressionistic methods of observing behavior were still in vogue and were employed by Loeb in reaching his conclusions sum-

marized above. A psychologist, Shepherd Ivory Franz, first introduced objective methods for assessing the effects of cerebral lesions in cats trained preoperatively on specific tasks and tested postoperatively. His monograph, *On the Functions of the Cerebrum: The Frontal Lobes*, was published in the *Archives of Psychology* in 1907 (150). His findings were largely overlooked by physiologists, however, and the possibilities of his methodological contribution lay unexplored until his associate, K. S. Lashley, adapted and extended the technique for his explorations of the rodent brain.

Lashley (286), perhaps more than any other investigator, carried the holistic view into its present controversial position. In addition to his contributions to theory, he presented a fourfold advance in methodology: (1) he adapted and developed objective indicators, performance on which could be expressed to considerable completeness in quantitative terms; (2) he used a sufficient number of experimental subjects (rodents) to permit statistical treatment of the results; (3) he employed the procedure of training–operation–critical testing and appropriate variants with his animals; and (4) he secured a quantitative index of the extent of the lesion in each brain by histological analysis. Using mazes of graded difficulty as indicators—on the grounds that "its formation [maze habit] involves processes which are characteristic of intelligent behavior"—Lashley found that (*a*) the capacity to form maze habits is reduced by destruction of cerebral tissue as is the retention of a maze habit established prior to a cerebral lesion, (*b*) in either instance the disturbance of the habit in quantitative terms is proportionate to the amount of cortical destruction (cf. Goltz, 182), (*c*) the same retardation in learning is produced by equal amounts of destruction in any of the cytoarchitectural fields of the cortex, and (*d*) the more complex the problem to be learned, the greater the retardation produced by any given extent of cortical lesion. Needless to say, these results dealt a severe blow to proponents of a strict localization view of intelligence. They pointed, however, toward one of the various psychological theories of intelligence stated earlier. In Lashley's own words:

> The results of the present experiments lend support to the theory which conceives intelligence as a general capacity, in the same measure that they oppose theories of restricted reflex conduction. The capacity to form and to retain a variety of maze habits and other less well-defined habits seems relatively constant for each individual, dependent upon the absolute quantity of cortical tissue functional and independent of any qualitative differentiation of the cortex or sensorimotor peculiarities of the problems solved. There is an indication that difficult tasks become disproportionately more difficult with decreased cerebral efficiency. Such facts can only be interpreted as indicating the existence of some dynamic function of the cortex which is not differentiated with respect to single capacities but is generally effective for a number to

which identical neural elements cannot be ascribed. In this there is close harmony with theories of a general factor determining efficiency in a variety of activities [p. 173].

2. *The aggregation theory.*—Whereas the holistic conception of intelligence implies a property of the cortex which is continuously distributed throughout the cortex, the aggregation theory conceives of this property as discretely distributed. Localized sensory fields for vision, audition, touch, etc., are bound together by manifold interconnections, the aggregate functioning of which gives rise to intelligence. Munk (328) suggested this view in 1890 and elaborated it in 1909. Munk's view is perhaps most closely paralleled by the multiple-factor theory proposed by Thurstone (398) and independently by Thorndike (397). It is primarily a sensory theory of intelligence and has been criticized for its failure to take into account the motor components of intelligence. It should be noted that Munk's conception is compatible with the quantitative results obtained by Lashley (286) where the degree of retardation was found to be proportionate to the extent of the cortical lesion; for as more and more sense fields are invaded by the lesion, the deficit in behavior might become increasingly large. Crucial experimental data on this point for any form other than the rat are still lacking.

Von Monakow (317, 318) cited his own experiments in support of those of Munk and in general concurred with the aggregation theory proposed by Munk. He deplored Munk's failure to give adequate weight to the motor components of intelligence. He regarded intelligence as involving the co-ordination of many diverse sensory and motor fields discretely distributed throughout the cortex. He emphasized the importance of distinguishing between the acute or transitory symptoms following a cerebral lesion and the residual or permanent effects of the lesion. He proposed his well-known theory of "diaschisis" (functional dissolution of related components) to account for the former, which, he believed, were responsible for a spurious emphasis toward specific localization of intelligence in the interpretations of many investigators. Using impressionistic methods for the observation of behavior, Von Monakow failed to find evidence of alteration in psychic behavior following removal of both frontal lobes in dogs and in macaque monkeys.

The aggregation theory has not lacked support from studies of human brain injuries. Kleist (267, 268) has presented evidence from an imposing group of over three hundred cases of gunshot brain-injured soldiers from World War I. In approximately one-third of this group, the frontal lobes were primarily involved. Kleist concluded that the convex lateral surfaces of the frontal lobes were associated with intellectual and psychomotor

activities. The orbital surfaces were associated with affective responses, such as feeling tone, depression or elation (euphoria, *Witzelsucht*, puerility), and even "moral insanity."

Kleist fractionated the various intellectual functions in a fashion somewhat reminiscent of the phrenologists. His clinical tests were in many instances far too crude to yield reliable evidence of the aggregate type of distribution in the cortex which he postulated with elaborate maps. Furthermore, his case material is similar to that of Feuchtwanger and, hence, is subject to the same objections (128).

Kleist's theory is, strictly speaking, a modified aggregate–regional-localization theory, since the aggregate distribution of intellect is localized in the frontal lobes.

It is of interest that the signs of frontal-lobe damage—lack of initiative, disturbances in attention, impairment of thinking processes, and emotional anomalies—are commonly encountered in the posttraumatic syndrome or patient with closed-head injury. This fact has been brought out by Grünthal (184) in an interesting series of cases. Grünthal followed seventeen cases of posttraumatic brain injury over a period of several years, giving them repeated clinical examinations. Sixteen of these cases came to autopsy and Grünthal was able to secure histological evidence in support of his clinical findings. In the interim following their injury, these cases had acquired medical histories and multiple diagnoses of functional disorders, including compensation neurosis. In twelve verified cases of frontal-lobe damage, the presence of brain damage was in no instance taken into account in the diagnoses of psychogenic disorders. Grünthal followed Kleist in localizing the intellectual faculties and loss of initiative on the convex lateral surfaces of the frontal lobes. Emotional disturbances and psychopathic behavior, he believed, followed injury to the basal or ventral surfaces of the brain.

3. *Regional-localization theory.*—The notion of a localization of ideas in the form of a cortical mosaic was fairly well discredited by the end of the eighteenth century (cf. Fearing, 125), although some enthusiastic proponents of phrenology continued to expound this doctrine. Two events of the latter half of the nineteenth century gave impetus to the development of a conception of regional localization of function in the brain. One of these was the announcement by Paul Broca in 1861 of his discovery of a speech center at the base of the third frontal convolution, an area in the brain which has since come to bear his name. The other was the discovery nine years later of an electrically excitable motor area in the cortex by Fritsch and Hitzig (159).

Broca (55), a French surgeon, chanced to examine a patient with a gangrenous infection, who thirty years before had been admitted to the Bicetre Hospital for the insane, near Paris. His sole defect at the time of his admission to the hospital and for thirty years thereafter was that he was unable to talk. He was regarded as normal mentally and was able to communicate intelligently by means of signs. He came under Broca's care five days before his death. Broca ascertained that the requisite musculature for speech was physiologically intact, yet, nevertheless, the patient could not speak. Upon examination of the brain, Broca found a large area of softening in the left hemisphere extending from the frontal lobe back to the parieto-occipital juncture and downward to include the superior portion of the temporal lobe. Faced with this diffuse lesion, Broca produced a masterpiece of deduction. He reasoned that Gall and his followers in phrenology localized the power of speech in the frontal lobes; the only point at which the lesion before him involved the frontal lobe was at the base of the third frontal convolution; *ergo*, this was obviously the location of the center for speech!

Following his experiments on the electrically excitable motor areas of the cortex, Hitzig (230) proceeded to make an experimental attack upon the problem of localization of the higher processes in the brain. He made a crude beginning at observing his animals in standard situations both before and after their operation, but, on the whole, his behavioral methods were essentially impressionistic. His prestige as an investigator was great, however, and his findings in favor of a regional localization of intelligence in the frontal lobes became a standard reference in refuting the views of Goltz and of Munk. He wrote:

> I adhere today to the hypothesis I put forward in 1870 that the cortical centers are merely collecting centers. I can support the opinion, which has often been expressed, that deep or very extensive lesions affecting the central mechanism necessarily interrupt a multitude of bundles which unite different cortical regions of the brain with one another, and must, consequently, give rise to symptoms that are susceptible to a relatively rapid improvement. To this category belong the transitory disturbances of sight which follow deep lesions affecting different regions of the hemispheres. I find myself in opposition to Munk as regards the nature of the higher intellectual functions and their relations with the anatomical substratum. Munk holds that there are no such things as special organs for these functions and that there is no necessity for them. I believe with him that intelligence, or more correctly, the store of ideas, is to be sought for in all parts of the cortex, or rather, in all parts of the brain; but I hold that abstract thought must of necessity require particular organs and those I find in the frontal brain [p. 261].

Hitzig thus gave an early expression to the unit theory of intelligence and assigned it a regional localization. The capacity for abstract thought is

the working conception of intelligence employed by Terman (393) as the basis for his revision of the Binet scales for the measurement of intelligence. In this theoretical juxtaposition of neurologist and psychologist was born the possibility of a crucial test of a specific hypothesis in human subjects. On the assumption that Hitzig and Terman entertained the same notion of intelligence and that Terman had produced a valid measure of the capacity for abstract thought in the Stanford-Binet test, it but remained to apply this scale to patients before and after frontal-lobe operations in order to secure a quantitative index of the resulting dementia. As we shall see later, this procedure has been carried out in neurosurgical patients. However, quantitative signs of dementia have, in some instances, failed to appear. In fact, the results of these procedures to date are such that it has been impossible to interpret with certainty the point at which the invalidity arises: in Hitzig's localization in the frontal lobes of something which he called intelligence, or in Terman's measuring device by which he purports to measure something which he calls intelligence. This impasse clearly points up the urgent necessity of clarifying the nature of intelligence.

Paul Flechsig (137) approached the problem of localization of function from a histomorphological standpoint. He divided the cortex into primary and secondary projection centers and association centers. The most important of the association centers in mental life, he believed, was the great posterior or parietotemporo-occipital associative zone, bounded in front by the postrolandic convolution and the insula and inferiorly by the hippocampus. Lesions occurring in this zone, according to Flechsig, produced dementia, while lesions in the frontal association areas did not. It should be noted that this zone includes most of Wernicke's language area, regarded by many neurologists as the region responsible for sensory aphasia. Examinations of patients with sensory aphasia have convinced me that seldom is one able to assess the degree of dementia in such cases with any reasonable degree of certainty. In comparison with the alterations in behavior to be noted clinically in patients with frontal-lobe lesions, the symptoms presented by the patient with sensory aphasia may be striking and yet may be easily mistaken for dementia or psychosis. It is likely that Flechsig fell victim to this possible error.

In general, the major contributions to the problem of intelligence by neurologists and physiologists have been based upon impressionistic methods for studying behavior and came largely during the latter half of the nineteenth century. They thus preceded or were independent of modern developments in neurophysiological techniques and conceptions,

"intelligence" tests and objective methods for studying animal behavior, and modern insights concerning the neurological-like character and widespread distribution of mental disorders.

Franz (150), an experimental psychologist, who worked variously in the Harvard Medical School, the Dartmouth Medical School, and the McLean Hospital in Boston, recognized the need for objective methods for studying behavior in investigations dealing with localization of function. Using puzzle boxes of the type developed by Lloyd Morgan and by Thorndike, Franz studied the acquisition and retention of a simple "escape" habit in cats and in monkeys. He trained his animals to a criterion of success before cutting the connections to the frontal lobes or to other parts of the brain. He concluded:

> When the frontal lobes are destroyed, recently formed habits are lost . . . . the loss is not brought about by lesions of other portions of the brain. The loss of associations is not due to shock, for lesions of other parts of the brain are not followed by loss of habit nor does the anesthetic and loss of food, etc., produce loss of associations. Unilateral lesions of the frontal areas are not followed by a loss, but there may be a slowing or retardation of the motor response. Habits once lost after removal of the frontals may be relearned. The relearning takes about as long a time as if the animal were learning a new association. Only newly formed habits seemed to be lost after such lesions. Long standing habits seemed to be retained. The emotional condition of the animal remains the same after as before the removal of the frontals [p. 63].

Franz also emphasized the subtle character of the changes following frontal-lobe damage:

> The experiments show not only that there are changes coincident with lesions or disturbances of the frontal lobes but that the associational loss is one that could not be determined by simple observational methods. The results suggest, furthermore, that for the determination of functional changes in man consecutive to and resulting from injuries to the associational areas accurate physiological and psychological methods should be employed [p. 63].

Thus did Franz become a proponent of regional localization of "associational processes"—he avoided the problem of defining intelligence by not using the term. His experimental findings tended to support the views of Hitzig and to discredit those of Flechsig. His finding that "the emotional condition of the animal remains the same after as before the removal of the frontals" was contrary to the experience of the Italian physiologist, Bianchi, who worked with dogs, foxes, and monkeys.

Bianchi (36) summarized the results of his experimental program on localization of function which dated back to 1883. He was an early proponent of regional localization of intelligence in the frontal lobes. He wrote able polemics in support of his views, although his experiments on dogs, foxes, and monkeys left much to be desired in the way of rigorous con-

trols. Nevertheless, the gradual surrender of most of his initial position by Flechsig, who had been an ardent supporter of the phrenological doctrines of Gall, in favor of localization in the frontal lobes was, in large measure, due to Bianchi.

From his first experiments on dogs, Bianchi concluded:

> *Unilateral* destruction of the prefrontal lobe of the dog was not followed by any noteworthy symptom, whilst *bilateral* destruction brought into evidence a distinct alteration of character, marked especially by weakening of all the psychic manifestations: defect in perceptive judgment; exaggerated fear, resulting from defective critical capacity and inability to make use of the physical powers although these were well preserved; amnesia and, generally speaking, a psychically blind behavior; defect in initiative and resource; lack of finality in the complex movements, as betrayed by incoherent conduct and diminished vivacity (lowering of the psychic tone). In fact, any ordinary individual would have classed these dogs as weak-minded [p. 71].

Without specifically defining intelligence, Bianchi, nevertheless, stated his conception in the form of a question:

> Does there exist a cerebral organ which has the faculty of utilizing the mental products of the sensory areas of the cortex for the construction of mental syntheses more suited for the spiritualization, and, hence, the cognition, of nature, an organ giving rise to reactions upon the world which, on the basis of individual and collective experience, permit a higher adaptation of the individual to his physical and social environment, an organ which renders possible the unfolding of a long process of logically connected thought [p. 73]?

Bianchi maintained that the frontal lobes were such an "organ of intellect" and that they controlled the highest levels of integration.

IMPRESSIONISTIC VERSUS QUANTITATIVE METHODS

Except for the studies by Franz (150) and by Lashley (286), impressionistic methods of observing behavior were the basis of the extended controversies concerning the localization of intelligence in the brain. To be sure, clinical observations on human cases had been used to support the evidence secured by extirpation experiments on infrahuman animals, but such evidence had been mobilized equally by proponents for the holistic view (Goltz, Ferrier, Loeb, Feuchtwanger, Lashley), for the aggregation view (Munk, Von Monakow, Kleist), and for the regional-localization view (Broca, Hitzig, Flechsig, Franz, Bianchi) and hence scarcely could be regarded as a valid test of any hypothesis. Perhaps more noteworthy is the fact that Franz and Lashley, using quantitative methods, reached such divergent conclusions. Lashley, working with rodents, it will be recalled, concluded in favor of the holistic view, while Franz, working with cats and monkeys, concluded in favor of regional localization. Lashley (286), in refuting Franz's and Bianchi's conclusions, stated: "The obser-

vations of Franz upon the loss of habits following destruction of the frontal lobes shows a localization of habits within this area; but, since his animals relearned the problems *without significant retardation,*[1] the results are directly opposed to those of Bianchi and indicate that the animals are not demented after destruction of the frontal lobes" (p. 9).

Franz's conclusion concerning the relearning of habits lost by frontal-lobe damage was to the effect that "the relearning takes about as long a time as if the animal were learning a new association." In other words, the traces of initial learning were lost following the operation, and the animal was reduced to its prelearning level of performance. The amnesia produced by damage to the frontal lobes undoubtedly represents a radical alteration in behavior (see Franz's Cases X, XXII, and XXIII [150, pp. 59–60]), since trace systems for simple habits are normally stable and are possibly never eliminated by passage of time alone. The possibility must be considered that the cat and the monkey present more highly differentiated brains than does the rodent and that holistic principles which hold for the one give way to regional localization in the higher forms, including man. But let us turn to the present studies for quantitative evidence bearing on this problem.

[1] Italics mine.

CHAPTER V

PRESENT STUDIES

THE quantitative findings reported in this investigation are based upon the test results from a battery of twenty-seven neuro-psychological indicators applied individually to two hundred and thirty-seven subjects. Supplementary observations are reported for approximately thirty additional individuals. Administering and scoring these tests averaged about fifteen hours per subject. Each subject participated in the study voluntarily without remuneration.

Classes of experimental subjects.—The distribution of the various categories of subjects examined is shown in Table 1. A brief description and case number for each of the fifty cerebral lobectomies and diagrams of the brain lesions are given in Appendixes A and B.

Background of the present studies.—Developments in neurosurgery during the last two decades have yielded an increasing number of co-operative patients with circumscribed brain lesions, in some instances involving total removal of certain physiological areas in the cortex (191, 206). From neurological, psychiatric, and psychological points of view, such material stands in marked contrast to the head-injury cases produced by World War I, which, in the opinion of such contemporaneous experts as Forster (144) and Von Monakow (318), were wholly unsuitable for studies of localization of function.

The improvement in neurosurgical techniques revived medical interest in the nature of the deterioration, if any, observed after surgical removal of various parts of the brain. In this country, Dandy (97), as early as 1925, on the basis of cursory clinical examinations on a man in whom he had removed the left prefrontal lobe, concluded that no appreciable mental disturbances were produced by the lesion. Only eight years later, Bailey (8), a pupil of Cushing, wrote: "I hesitate before amputating a frontal lobe. This procedure is always followed by more or less great alteration in character and defects in judgment. In a washerwoman these results may be of little concern, but when the patient is a professional business man, who must make decisions affecting many people, these results may be disastrous" (p. 433). Such divergent views are strikingly reminiscent of those expressed by Munk, Goltz, Loeb, and others at the beginning

of this century. Thus, the first promise of modern neurosurgery to throw light on the problem of localization of mental functions threatened at the outset to be evanescent. It followed, of course, that a solution of many related problems, such as the contributions of the nervous system to neurosis and schizophrenia, would be postponed. Similarly, the ancient philosophical controversies over the "mind-body problem," currently finding expression in modern medicine under the heading of psychosomatics, would find no resolution from this source.

In the face of an increasing supply of suitable neurological material for study, in the form of neurosurgical patients with circumscribed lesions of the brain, it seemed desirable to attempt to develop behavioral methodology which would extend our knowledge of principles of localization of

TABLE 1

DISTRIBUTION OF EXPERIMENTAL SUBJECTS

Control subjects	30
Cerebral lobectomies	50
General medical patients (military head injuries)	63
Neuropsychiatric patients (military head injuries)	57
Civilian patients (closed-head injuries)	28
Pre- and postlobotomies (subcortical lesions)	9
Total	237

function in the brain. In 1935 a special laboratory was established for the study of neurological patients from the service of Dr. Percival Bailey at the University of Chicago Clinics. Dr. Bailey and his associates, Dr. Paul Bucy and Dr. A. Earl Walker, and other medical specialists adopted the practice of referring cases suitable for psychological and physiological studies to this laboratory. It should be emphasized that for perhaps the first time in the history of modern neurosurgery, a group of neurologists and neurosurgeons have fully co-operated in making available their cases for careful study by experimental methods. It is the writer's belief, supported by the national and international reputations of these men, that this neurological material represents the application of the highest standards of modern neurological and neurosurgical skill. If, in the present studies, qualitative and quantitative evidence has emerged which points to certain forms of localization and to special forms of behavioral deterioration, such findings can but affirm the generally high quality of the material.

In several instances it has been possible to make preoperative "base-line" studies as well as extensive postoperative studies. In selecting patients for prolonged investigation, the question of preoperative findings has not been made a limiting consideration, however. This is especially true where there was a possibility of securing a satisfactory preoperative history, including a qualitative appraisal of the psychological adequacy of the individual personality. In general, individuals were chosen who presented minimal clinical and sociological signs of an intracranial lesion. In one case, through those increasingly rare accidents of diagnosis, it was possible to make pre- and postoperative studies on a patient who, prior to right frontal lobectomy, probably had an essentially normal brain. Many of the patients reported here have been under study for a period of nearly ten years. Most of them are gainfully employed and have otherwise made a reasonably adequate social adjustment. Others have died, and in some cases it has been possible to make detailed histological studies of the brain lesion (193).

Qualitative study of individual patients.—The propaedeutic or preliminary phase of any science involves a general survey or observation of individual events (271, 280, 343, 428). This appears to be a necessary prelude to the isolation of those phenomena in nature characterized by certain constancies which constitute the parsimonious descriptions of science. It is in this stage that the investigator formulates the working hypotheses concerning where to search which logically precede the adoption of particular methods of search (343). These considerations are especially applicable in the study of psychopathology, in which the tendencies are strong to carry over a priori concepts and methods from the field of normal behavior. To obviate this potential source of error, the writer early in the present program confined his efforts to the detailed study of a few, carefully selected, patients with circumscribed brain lesions (191, 193, 205). Every effort was made over a period of months or years to explore any avenue which might lead to a total appraisal of the personality and social adequacy of the individual case. Much of this information was collected in a systematic fashion during repeated examinations in the laboratory. Equally relevant and useful observations were made as opportunities arose in social situations outside the laboratory. In general, the backlog of information concerning this small group of patients was drawn from the following sources:

1. Behavior of the patient in a wide range of laboratory "stress" situations over a period of months or years following a brain operation. Occasionally comparable studies were made prior to, as well as after, the operation.

2. Consultations with clinical neurologists concerning the postoperative neurological status of the patient.

3. Consultations with psychiatrists and psychoanalysts, in some instances involving appraisal of the effects of brief psychotherapy.

4. Consultations with other medical specialists when acute illness developed.

5. Consultations with psychiatric and medical social workers concerning the adjustments to family and to work of certain patients.

6. Consultations for routine psychometric examinations and for survey of vocational aptitudes.

7. Social correspondence in the form of letters and telephone conversations exchanged between the writer and individual patients.

8. Personal diaries kept at the suggestion of the writer. In one case this involved a complete daily diary covering the first year following total occipital lobectomy.

9. Where possible without prejudice to the patient, consultations with family, business, and personal associates.

10. Upon the death of certain patients, consultations with life insurance companies.

11. Phonographic recordings of interviews, in some instances secured by means of concealed-microphone technique.

12. Motion pictures of laboratory and social behavior, in some instances secured by means of concealed-camera technique.

13. Examination of school records.

14. Social visits to the home by the writer or by his assistants.

15. Observations by the writer of the behavior of certain patients in such social situations as eating in clubs and restaurants, attending a motion-picture theater, attending athletic events, and exchanging holiday greetings. A substantial part of the background information is thus of the "clinical" variety. This body of material is too voluminous for inclusion here. It could properly constitute the basis for another communication.

Quantitative study of individual patients.—The task of securing comparable quantitative data for any considerable number of indicators on patients in a general hospital setting is not a simple one. Considerations of general health, the presence of special pathologies (e.g., sensory or motor), availability of the patient for complete examinations, fluctuations in the attitude of the patient toward being examined, the introduction of new indicators into the battery, and instrument failure are all factors which retard the accumulation of a significant body of data. In the initial phases of the study all examinations were carried out by the writer. It was obviously preferable to train personnel to make the various measurements and to establish the procedures on a truly objective basis. It proved possible to do this. Accordingly, the quantitative data reported here are based upon normal individuals and patients selected by the writer in consultation with medical experts but are otherwise completely free from this investigator's personal bias. In addition to its possible significance for the field of psychology and for various special medical disciplines, this study is offered as a contribution to quantitative biology.

The problem of satisfactory control studies.—The problem of satisfactory control observations becomes extremely complicated in developing neuropsychological indicators. It is desirable that the investigator in this field keep "normality" in mind at all stages in the development of his instruments. On the other hand, ultimate statistical validation in terms of an adequate random sample of the general population should come at the final stage of development rather than at the beginning. One reason for this is that otherwise any further alteration of the indicator may destroy its statistical validity with reference to groups of normal individuals. Another, perhaps less obvious reason, is that only by the detailed study of pathological forms of behavior is it possible to know what behavioral controls are necessary. It seems clear that in many types of investigation of behavior an "adequate random sample of the general population" can be defined only in terms of what other controls can be or have been instituted. There can be little doubt that failure to understand this point has resulted in much confusion, even in those few instances where experimental studies have been attempted in this field.

By way of illustration of the problem of suitable controls, we may refer to a study of frontal-lobe functions by the psychiatrist Rylander (354). This investigator chose for study, by means of an assortment of psychological and clinical tests, a group of patients with frontal-lobe lesions. As a control group, he employed a similar number of "normal" individuals, each of whom was approximately matched with an operated subject in certain known ways. This technique of paired subjects is commonly employed in biological investigations of infrahuman animals and is justified where control of a large number of variables, as in the case of litter mates, is possible. Where such is not the case, and this includes most studies involving man, its use may yield a spurious picture of normal variance. Perhaps even more serious is the fact that its use may lead to the conclusion that adequate controls have been employed (cf. Hebb, 219). While Rylander's conclusions are all stated in terms of functions of the frontal lobes, his failure to include a single patient with a primary lesion outside the frontal lobes makes it impossible for him to define the brain-injury factor under study as "frontal lobe." Each of Rylander's frontal-lobe patients differed from its proximate control subject in an important additional fact that there was a history of an "operation on the head." Such an operation involves shaving of the scalp and at least temporary disturbance of the schema of the head for the patient. These events not uncommonly result in mild or severe psychic trauma which may or may not be associated with the appearance of transitory or persistent neurotic

symptoms. Such neurotic symptoms are also seen in patients with frank psychoneurosis but without brain lesions demonstrable by present-day methods. Had Rylander seen fit to include a few psychoneurotic patients in his study, it would have been possible to estimate the probable effect of this factor upon performance in the tests which he employed. It is indeed unfortunate that this potentially valuable study thus remains indeterminate in spite of the inclusion of a control group of normal individuals.

The principle of blended samples.—A common procedure for insuring the inclusion of several degrees of freedom for variance in a control group is to employ a random sample of the general population. To do this in adequate numbers frequently leaves the investigator little time for further study of pathology. In the present study it was imperative that the factor of any possible brain injury be excluded from the control group. In order to provide guaranties of this, it was necessary to secure a relatively detailed medical history for each individual. Obviously, this is more readily done with a small than with a large group of subjects. To obtain the advantages of a small group, and at the same time the multiple degrees of freedom for variance offered by a large group, it was decided to blend the control group along the lines of maximal heterogeneity.[1] Accordingly, various clusters or constellations of psychological and sociological data (as encountered in the patients studied) have been taken as the reference point of normality (see Table 2). In establishing the limits from within which the clusters were drawn, a roughly empirical notion of two standard deviations of the general population has been kept in mind. Thus, for example, it was assumed that two standard deviations of the age distribution of the general population in the United States would fall well within the limits of one year and seventy years. Likewise, it was assumed that two sigma for the distribution for years of schooling would fall within the limits of one year and twenty years; for annual incomes, between the limits of $500 and $6,000, etc. In terms of the behavioral indicators which have been employed in these studies, it is believed that an adequate random sample of the general population would have approximately the same degrees of freedom for variance as the plausible intercolumn combinations. The composition of the control group employed is shown by the items in boldface type of Table 2. For convenience, each control subject has been assigned a Roman numeral while each experimental subject has been assigned an Arabic numeral.

Normal control subjects.—Subjects I–XIV were civilian control sub-

[1] The principle of heterogeneity in the general theory of measurement is succinctly discussed in R. A. Fisher's The design of experiments (134).

jects. As a group, they ranged in age from fourteen to fifty and averaged twenty-six years. Each was induced to take the battery of tests without compensation. Nine of the group were males, and five were females. Detailed inquiry failed to reveal any history of actual or probable exposure to the possibility of brain injury, traumatic or otherwise. It should be mentioned that all the male subjects under thirty years of age were faced with the prospect of early induction into the armed services. In the case of

TABLE 2

SHOWING HETEROGENEOUS COMPOSITION OF CONTROL GROUP*

Age	Education	Extraction	Religion	Occupation	Income	I.Q.
70	20	**American**	**Agnostic**	Accountant	**$10,000**	150
65	19	**Balkan**	**Atheist**	**Artist**	9,500	145
60	**18**	Chinese	**Baptist**	Business executive	9,000	**140**
55	**17**	Dutch	**Catholic**	Clergyman	8,500	**135**
50	**16**	**English**	**Disciples**	Draftsman	8,000	**130**
45	**15**	**French**	**Episcopalian**	Engineer	7,500	**125**
40	**14**	**German**	Friends	**Entertainer**	7,000	**120**
35	**13**	Greek	**Hebrew**	**Farmer**	6,500	**115**
30	**12**	**Irish**	**Lutheran**	**Housewife**	**6,000**	**110**
25	**11**	Italian	**Methodist**	**Laborer, semiskilled**	5,500	**105**
20	**10**	Japanese	None	**Laborer, unskilled**	5,000	100
15	**9**	Mexican	**Presbyterian**	Merchant	4,500	**95**
10	**8**	Negro	**Unitarian**	Nurse	4,000	90
5	**7**	**Polish**	**Science**	Politician	3,500	**85**
	6	Russian		**Professional**	**3,000**	80
	5	**Scandinavian**		Professor	**2,500**	**75**
	4	**Scotch**		**Secretary**	**2,000**	**70**
	3	Spanish		Social worker	**1,500**	65
	2			**Teacher**	**1,000**	60
	1			**Technician**	**500**	55
	0			**Trade**	**000**	50
				Student	000	45

* Items in boldface type are present in control group employed.

Subject IV, it is believed that this fact provided the basis for a mild clinical depression. Mild degrees of acute anxiety were present in Subjects XII and XIII, youngest and oldest, respectively, in the group. The others appeared relaxed and well motivated during the taking of the various tests. All the subjects were regarded at the time of examination as having normal personalities and as being reasonably well adjusted. Support for this interpretation was afforded by the various quantitative measures of personality functions employed as part of the general investigation.

Military controls.—Subjects XV–XXIV were members of the armed services with psychiatric diagnoses of mild psychoneurosis. They ranged

in age from twenty-two to thirty-eight and averaged twenty-nine years. Some of them had had military combat experience, but detailed inquiry revealed no history of head injury. Their attitudes toward the various tests were, in general, those of interest and co-operation. Several gave abnormal scores on the personality tests, thus supporting the psychiatric diagnoses. Their symptoms at the time of testing ranged from mild to severe headaches; from loss of appetite, easy fatigability, acute or chronic gastrointestinal disturbance to insomnia and minor disturbances in memory functions. Inclusion of these subjects in this group serves to control the factor of armed-service-connected attitudes toward personal illness and toward being tested. Both of these considerations are of importance in evaluating the quantitative results for other members of the armed services, as presented in subsequent sections.

Miscellaneous controls.—Subjects XXV–XXX constituted a miscellaneous group of individuals who were under some known psychological or biological stress at the time of examination. Subject XXV, for example, was a military prisoner at the time of testing. He had been found guilty of high crimes by court-martial proceedings and faced imminent sentence, either life-imprisonment or execution. Although co-operative, he was obviously depressed in mood while taking the tests. Subject XXVI was a thirty-three-year-old female with a severe nonagitated depression, with associated homicidal impulses which were directed against her child. She was examined prior to undergoing an operation known as "lobotomy" for relief of her condition. Subject XXVII was a thirty-nine-year-old woman who had been committed to a state mental institution because of suicidal and homicidal impulses. She was examined prior to undergoing lobotomy. Two months after her brain operation she was re-examined. The results of this second examination are listed under Subject XXVIII. Subject XXIX was a twenty-eight-year-old male who had been given intensive psychotherapy for relief of strong suicidal impulses and relatively strong homosexual trends in his personality. He was examined prior to lobotomy and again two months later. The results of the second examination are listed under Subject XXX.

From the descriptions of the control subjects given, it will be apparent that several degrees of freedom for variance in addition to those included in Table 2 are represented in the control group.

CHAPTER VI

QUANTITATIVE RESULTS

OUR first task is to establish a rational hypothesis as to the kind and number of factors involved in biological intelligence. We have seen that previous attempts to relate human intelligence to brain functions have failed, in part because of insufficient attention to this task. In so far as possible, our objective in this chapter will be a definition of biological intelligence in operational terms. The specific points which we must consider concern (*a*) selection of a battery of behavioral indicators, (*b*) selection of a group of subjects for examination by this battery, (*c*) examination of the quantitative relations between performance on the various tests, and (*d*) isolation by quantitative methods of the principal factors involved in the tests.

Total battery of tests employed.—The total battery of tests employed in these studies consists of twenty-seven behavioral indicators, which are described briefly in Appendix C. In setting up the test battery an effort was made to include a wide range and variety of items, including (*a*) tests which in preliminary studies had shown promise of differentiating between brain-injured and normal individuals, (*b*) tests of psychometric intelligence, (*c*) tests of various personality functions, and (*d*) tests of various sensory capacities, such as acuity of vision, hearing, and color vision. Perhaps the most important consideration which determined the final composition of the battery was the criterion that the battery must be applicable without essential alteration of instructions or technique to normal control individuals and to brain-injured and psychiatric patients serving voluntarily as subjects.

Selection of experimental subjects.—The decision concerning an appropriate sample population of subjects on which to base our analysis of biological intelligence involved several considerations. In the first place, it was desirable that the subjects chosen be essentially normal individuals and yet be comparable in many ways with our neurosurgical and psychiatric patients. The group finally chosen for this section of our studies consisted of fifty healthy adult males, drawn from the head-injury service of the Gardiner Hospital, an army general hospital in Chicago. All these cases were regarded as medically recovered from a recent concussive type

of head injury. All had experienced an interval of unconsciousness of varying duration up to one hour at the time of injury. Detailed neurological and psychiatric examinations, including detailed sensory examinations, and complete medical histories were available for each. Each case was tested individually on two successive days in the writer's laboratory in Billings Hospital of the University of Chicago, under good test conditions.

Subbattery of tests for correlation analysis.—From the total battery of twenty-seven tests, those tests were selected which yielded objective scores suitable for treatment by means of the Pearson coefficient of correlation, r, and which seemed likely to reflect some component of biological intelligence. Thirteen tests for the subbattery were thus finally selected. They are identified by an Arabic numeral and by name in the list below. One or both of these identifying signs is used in the various tables of data which follow.

SUBBATTERY OF TESTS

1. *Carl Hollow-Square Performance Test for Intelligence.*—A nonverbal test for psychometric intelligence, which yields an I.Q.

2. *Halstead Category Test.*—A nonverbal test involving visually induced abstractions of size, shape, color, etc., presented by means of a multiple-choice projection apparatus.

3. *Halstead Flicker-Fusion Test.*—A nonverbal test for low-level critical-fusion frequency.

✓4. *Halstead Tactual-Performance Test (Speed).*—A modification of the Seguin-Goddard form board, presented nonvisually for three timed trials.

✓5. *Halstead Tactual-Performance Test (Recall).*—A drawing (recall) test for incidental learning induced by Test 4.

✓6. *Halstead Tactual-Performance Test (Localization).*—A drawing (localization) test for incidental learning induced by Test 4.

9. *Henmon-Nelson Tests of Mental Ability.*—Paper-and-pencil tests for psychometric intelligence, which yield an I.Q.

13. *Speech-Sounds Perception Test.*—A multiple-choice speech-sounds discrimination test presented from a phonograph record.

✓14. *Halstead Finger-Oscillation Test.*—Rate of activation of a mechanical counter with the extended index finger.

✓16. *Halstead Time-Sense Test (Memory).*—Memory for a ten-second interval is tested under nonvisual conditions by means of an electric clock following twenty preliminary trials with vision permitted.

✓17. *Halstead Dynamic Visual Field Test (Central Form).*—A visual-

discrimination test for simple geometric forms differing in color, exposed at the foveae for twenty milliseconds along with a graduated peripheral target in some exposures.

18. *Halstead Dynamic Visual Field Test (Central Color).*—A visual-discrimination test for the color component presented as part of Test 17.

19. *Halstead Dynamic Visual Field Test (Peripheral Component).*—A threshold test for peripheral vision, presented as the peripheral-target component of Test 17.

Table of intercorrelations of the test scores.—The seventy-eight intercorrelations between the scores for the thirteen indicators obtained from our group of fifty subjects are presented in Table 3. Each indicator is

TABLE 3

TABLE OF INTERCORRELATIONS FOR THIRTEEN NEUROPSYCHOLOGICAL TESTS

Test	1	2	3	4	5	6	9	13	14	16	17	18
1												
2	.503											
3	.059	.084										
4	.187	.249	.270									
5	.299	.432	.287	.196								
6	.253	.480	.239	.431	.444							
9	.313	.528	.117	.273	.377	.414						
13	.086	.343	.148	.345	.192	.192	.412					
14	.002	.334	.326	.303	.130	.402	.323	.369				
16	.254	.320	.173	.221	.044	.223	.368	.238	.267			
17	.136	.262	.413	.214	.350	.378	.412	.428	.501	.360		
18	.050	.329	.435	.200	.242	.295	.307	.421	.459	.242	.809	
19	.124	.144	.026	.363	.255	.250	.193	.036	.005	.062	.058	.026

identified by its Arabic numeral assigned in the above list and in Appendix C. The correlation coefficients were computed twice from Holzinger correlation sheets to insure accuracy. It may be noted that the intercorrelations are all positive in sign and range from 0 to .809.

Application of factor analysis.—Factor analysis is a mathematic technique for examining a table of intercorrelations and reducing or attributing its variance to a relatively small number of factors (237). In this sense, it yields a parsimonious description of the variance obtained from a group of test scores. This is extremely advantageous where, as in the present instance, it is probable that some of our tests are measuring the same functions but in varying degree.

Copies of our table of intercorrelations were supplied to Professor Karl Holzinger and to Professor L. L. Thurstone. They have separately and independently analyzed the coefficients by somewhat different techniques

of factor analysis. Each has made his analysis on a "blind" basis in the sense that he worked without firsthand knowledge of the variables involved. Professor Holzinger has supplied two factor solutions for our problem. One is an orthogonal solution similar to that employed by Spearman (378). The other is an oblique solution, somewhat similar to the technique employed by Thurstone (401). Both the Holzinger solutions yielded a four-factor description of the variance. These solutions were published by the writer in the spring of 1945 (199).

TABLE 4

OBLIQUE-FACTOR MATRIX (THURSTONE) FOR THIRTEEN NEUROPSYCHOLOGICAL INDICATORS

Indicator	Description	Factors			
		C	A	P	D
1	Carl hollow-square (I.Q.)	.25	.45	−.07	.04
2	Category	.49	.63	.09	−.03
3	Flicker fusion	.00	.04	.54	.05
4	Tactual performance speed	.12	−.02	.04	.61
5	Tactual incidental recall	−.02	.66	.43	−.02
6	Tactual incidental localization	.19	.34	.25	.29
9	Henmon-Nelson (I.Q.)	.58	.27	−.05	.23
13	Speech perception	.49	−.06	.06	.22
14	Finger oscillation	.40	−.06	.25	.18
16	Time-sense memory	.43	.11	.08	.02
17	D.V.F. form	.41	.07	.64	−.03
18	D.V.F. color	.41	−.03	.61	−.06
19	D.V.F. peripheral	−.15	.11	−.06	.54
		Σ 3.60	2.51	2.81	2.04

Thurstone has developed a technique of multiple-factor analysis wherein he rotates the reference axes until he finds the smallest number of orthogonal co-ordinate axes in terms of which he can express the major variance of the correlations (403). A general factor, in the sense of Spearman's G, does not appear in such solutions, although in certain types of problems a general factor may subsequently be extracted as a second-order factor (402). Thurstone has employed his method in isolating what he terms "primary mental abilities." He is interested in the psychological intelligibility of the factors isolated, whether they be general or specific in nature. Thurstone's method has undergone considerable refinement in recent years. It has thus far been used largely in the field of psychological testing, but there would seem to be no fundamental reason why it should not prove to be a very useful tool in several fields of biology and of medicine.

Professor Thurstone's oblique-factor matrix solution for our table of intercorrelations is presented in Table 4.

It may be noted that the variance of our table of seventy-eight intercorrelations is here distributed into four basic factors, indicated as columns C, A, P, and D in Table 4. The coefficients shown in each of these columns represent the factor loading of each test on each of the four factors, i.e., the extent to which any particular test is correlated with any given factor.

With our subbattery of tests now projected as a measuring device for four central factors, C, A, P, and D, for our group of subjects, our next task is to inquire to what extent these factors are psychologically meaningful. In other words, can we make an interpretation of each of the four factors which takes into account the nature of the task presented in each test as well as its high or low factor loading on the various factors. If so, then we will have approached our initial goal, which was to set up an operationally defined conception of biological intelligence in terms of the number and kind of factors involved. A further and crucial check on the validity of our basic factors will be afforded when, in Part II of this volume, we apply the same tests to individuals with focal brain damage and to normal control individuals.

CHAPTER VII

THE CENTRAL INTEGRATIVE FIELD FACTOR C

Everyday experience suggests that the quest of psychologists for "pure" measures of mental ability free from the influence of prior learning is likely to end in failure. The reason for this is that learning is itself an integrative process which bears many resemblances to behavior commonly associated with intelligence, such as thinking, judging, imagining, and the like. Fortunately for our purposes, studies of the mental growth of children cast some light on these relations. Even before the child has reached an age for formal schooling, myriad experiences have already registered and become integrated as a part of the reality-testing and reality-extending processes of a developing ego. His world of the senses is already well differentiated perceptually. Significant "signs" have become effective in influencing his behavior in much the same way as in his distant and less distant cousins, the rat (287) and monkey (271). But a world of symbols (of relations-among-relations) has only just begun to take form. As problem situations arise for him with their *ad hoc* rules and a priori standards to which he must conform, new realities emerge which are foreign to his world of senses. But he must garner from this new world of symbols a basis or framework of orientation or security. He must do this at the same time that he is extending his world of senses. The resultant of this dynamic competition is a matrix or ground process of the "old" against which the figured "new" may be tested without undue degrees of anxiety. Chaos is avoided because the dominant term in the prevailing psychophysical relation is stable. Stimuli in the outer world which once would have been explosions in his world of senses can now be tolerated because a new threshold of awareness has been established. The child has become less stimulus-bound in his motilities. A central integrative field has been differentiated from the products of a quasi-amorphous past. Upon the expansion of this ground function in the sense of Gestalt, possibly upon its ever-expandingness, is predicated the growth of adaptive intelligence. Here is the melting-pot of the old and the new wherein learning and intelligence coalesce.

In so far as the above outline of ego segregation fits the growing child, we should expect its processes to influence or to be reflected in our meas-

urements of biological intelligence. This is especially true where the available data are drawn, as in the instance of the present studies, from portions of the developmental curve bounded by early adolescence and by adulthood. Let us examine our factor pattern in Table 4, page 41, for such influences as may be reflected by our battery of indicators.

The factor loadings (entry for each test for each factor) under factor C given in Table 4 vary considerably from test to test. Of the thirteen tests, seven have substantial factor loadings on C of .40 or higher, while six have vanishingly small loadings in the neighborhood of zero. Is this dichotomous distribution compatible with our conception of a central integrative field factor?

INDICATORS WITH HIGH FACTOR LOADINGS ON C

First, let us consider those tests which have high factor loadings separately from those which have low factor loadings. Reading down column C we find that indicator No. 2 has a loading of .49; No. 9, .58; No. 13, .49; No. 14, .40; No. 16, .43; No. 17, .41; and No. 18, .41. Indicator No. 2 is the writer's category test. It consists of 336 items, the first 16 of which are warm-up items designed to establish a simple association between a Roman numeral presented on the projection screen and an identical numeral corresponding with each of four reaction keys. In this fore-period the subject learns that a correct response always and immediately rings a chime, whereas an incorrect response always and immediately elicits a rasping sound of a buzzer ("raspberry"). In the test proper, various categories of shape, size, position, number, color, fractions, and quadrants are made effective throughout a wide range of variation in content of the items. Some of the content is familiar to the subject (for example, solid or outline squares, circles, triangles, primary colors, letters of the English alphabet). The content of other items is unfamiliar, however (for example, nonsense figures comprised of interlacing lines, manikins, solid figures projected in space by broken contour lines, arbitrary designs accented in part by color or by shadings in brightness).

An item analysis of the errors made on this test by brain-injured and by normal individuals reveals an interesting fact. Whereas the normal individual threads his way through the various items, familiar and unfamiliar alike, grouping them readily into appropriate categories, patients with certain types of brain injury tend to adopt a priori sets or attitudes toward the items and especially toward items with unfamiliar content. These sets persist in the face of mounting contradictory evidence in the form of errors. Whereas one error is usually sufficient to point the normal

individual toward the correct hypothesis, an initial hypothesis adopted by a brain-injured individual may persist as an *idée fixe* through twenty or thirty errors or until help is given to avoid a catastrophic reaction. An illustration is afforded by one of the two quadrant subtests. A quadrant principle is made effective by the appearance in six successive items of three-fourths of an outlined square, each fourth of which contains a Roman numeral corresponding with one of the four reaction keys. In the first item, the upper left-hand quadrant with the Roman numeral I is missing, but the other three quadrants, numbered in clockwise fashion, are given as in

	II
IV	III

. In the second item, quadrant III is missing, but the other three are given, including quadrant I which was missing before. Item three is the same as item one. The fourth item presents quadrants I, II, and III, with IV missing from the lower left-hand position. The fifth item presents quadrants I, III, and IV, while the sixth item is the same as item four. To register a correct response in each instance, the subject must press the reaction key, the number of which is identical with that of the missing quadrant. After failing to grasp the principle involved, the brain-injured individual commonly reacts by stating that the "parts should be numbered this way" (i.e.,

I	II
III	IV

). He seems to approach these normally abstract figures through old learned habits of left-right, left-right sequences which are appropriate for reading the printed text of a page, for example, but which are inappropriate in the present setting requiring a left-right, right-left structuring of visual space.

That such failures are not specific for situations involving visual-spatial relations is attested, however, by the fact that the same brain-injured individual may decide arbitrarily that the color "blue" *must* mean the correct key or that the color "red" *must* mean the incorrect key regardless of the fact that he has had repeated experience with specific items to the contrary.

Similarly, the failures are not primarily on a perceptual basis since the patient may have no difficulty in describing accurately every detail of the item on the screen before him.

The basic difficulty in such failures appears to arise from disturbance of a central integrative process. Contemporary evidence from the senses loses out in the competition with altered ground functions of the individual. Affective accents are somehow displaced, with a result that tentative

propositions lose the character of tentativeness. The "as if" attitude gives way to the "is" proposition; "could be" manages to become "must be." As a result, total performance of the brain-injured on our category tests tends to be determined almost as much by the influence of a pathological central integrative process (C) as by a basic ability for abstraction of essential similarities and differences—or grouping to criterion.

Indicator No. 9 is one of the Henmon-Nelson Tests of Mental Ability. This is a paper-and-pencil test for psychometric intelligence which requires thirty minutes to administer. According to its authors, it has been found to correlate in the neighborhood of .80 with the revised Stanford-Binet scale. As may be seen in Table 4, it has the highest factor loading of any of our thirteen tests on our C factor, namely, .58. Analysis of its items reveals the following distribution: completion of numerical series, 12 items; word formation from scrambled letters, 4 items; vocabulary, 48 items; figural analogies, 9 items; sentence construction from scrambled words, 5 items; reading comprehension, 5 items; arithmetic computation, 5 items; and specific information, 1 item. With the possible exception of the completion of numerical-series items and the figural-analogies items, i.e., 21 out of a total of 90 items, this test obviously draws heavily upon specific learning of the individual. In the vocabulary items, which comprise more than half the test, skills are tapped which over a period of many years have had daily reinforcement. In so far as speed is a component in the total performance, it reflects the extent to which well-organized habits are touched by the trigger situations represented by the items of the test. Unlike our category test, this test requires little abstraction on the part of the subject, the major task of abstracting having been performed by the authors in constructing the test. The result of their efforts turns out to be a useful measure of our central integrative field factor C.

Indicator No. 13 is a speech-sounds discrimination test, made available to the writer through the courtesy of Professor Louis D. Goodfellow. It has a factor loading on C of .49. The test consists of 60 items composed of spoken sounds of nonsense syllables involving the digraph *ee*. The subject selects the syllable just heard from a group of four multiple-choice variants printed on a test blank. The critical sounds are presented from a phonograph record by a male voice.[1] The digraph *ee* is the middle part of every syllable spoken. Accurate performance is thus determined by the consonant or combination of consonants at the beginning of the spoken syl-

[1] We re-recorded the original test and used a professional male voice when early in our studies it was observed that the dulcet sounds of the female on the first recording seemed to be pleasantly distracting to certain of our military subjects.

lable and by the consonant or combination of consonants at the end of the syllable. The beginning consonants and their frequencies among the 60 items of the test are as follows: *Th*, 6; *W*, 4; *N*, 4; *H*, 4; *T*, 4; *L*, 3; *P*, 3; *S*, 3; *B*, 3; *D*, 3; *Y*, 3; *M*, 3; *K*, 3; *Sh*, 2; *F*, 2; *Fr*, 1; *Pl*, 1; *Wh*, 1; *Pr*, 1; *V*, 1; *Tr*, 1; *St*, 1; *Ch*, 1; *G*, 1; and *R*, 1. The ending consonants and their frequencies are: *T*, 8; *R*, 6; *N*, 5; *L*, 4; *M*, 4; *Z*, 3; *Ng*, 2; *D*, 2; *Nt*, 2; *Nd*, 2; *K*, 2; *V*, 2; *S*, 2; *Ts*, 1; *J*, 1; *St*, 1; *P*, 1; *Ch*, 1; *Nk*, 1; *Sh*, 1; *Kt*, 1; *Rz*, 1; *Th*, 1; *Ld*, 1; *B*, 1; *Ks*, 1; *Rd*, 1; *F*, 1; and *G*, 1. A total of twenty-five beginning and twenty-nine ending consonants or combinations of consonants is employed in varying the spoken syllables. On the printed record sheet checked by the subject, in each set of syllables (an item) one syllable corresponds with the one spoken, one has the beginning consonant or combination thereof plus the double vowel of the spoken syllable but a different ending, one has the ending consonant or combination thereof plus the double vowel of the spoken syllable but a different beginning, and the remaining one has only the double vowel in common with the spoken syllable. It is thus possible to analyze the errors made on this test as to whether they arise equally from the beginning and ending consonants of the spoken syllables.

An item analysis of the errors made by normal individuals and by brain-injured patients reveals that the former tend to make about the same percentage of beginning errors as of ending errors (48 and 52 per cent, respectively). Our brain-injured patients, on the other hand, tend to make about three times as many ending errors as beginning errors (76 and 24 per cent, respectively). This observation suggests that, as in the case of our category test, instabilities in the central integrative field (C) intrude upon perceptual processes that are initially "correct" but are disrupted before reaching completion. Closure is hastened following the initial clues and takes place without inclusion of critical ending clues.

Indicator No. 14 is the writer's finger-oscillation test. The subject is instructed to work the lever arm of a mechanical counter up and down "as fast as he can," using the index finger of the dominant hand. He is given five trials of ten seconds each. The downstrokes for each trial are recorded separately and then averaged for a final score. Normal individuals of both sexes, through several decades of age range, tend to average about five downstrokes per second. If we assume that about the same amount of time is consumed in the upstrokes (or even less, since mechanical impedance to upward motion is less), we approach a value of ten cycles per second as an average value. This may or may not be related to the fact that a rhythm of ten cycles per second is now generally regarded as

the most common alpha rhythm in the normal adult human electroencephalogram. Little is known as yet concerning the relation of motor speeds to brain rhythms, although it would not be surprising should a cyclic mechanism in the brain or cerebellum prove to have a direct relation.

It is possible that performance on our finger-oscillation test is limited by individual levels of aspiration for the task. The individual may respond to the instruction "as fast as you can" with an a priori ceiling which he adopts for himself before the first trial. Through the influence of old habits of motor discharge rate, this ceiling might be significantly below his potential motor speed on a new task. Two observations suggest that we might thus account for the factor loading of .40 which this test has on C. The first is that the highest oscillation rates made by any of our subjects have been made by trained speed typists. Yet there is no reason to believe that their general motivation for the task was greater than for other subjects. The second observation is that the awareness aspect of the task for the subject provides him with no reliable clue as to the level of his performance. His judgment either as to his fastest trial or as to his slowest trial in five is wholly unreliable. He may associate maximal effort on his part with either. Just as the rate at which an individual reads various types of prose tends to be determined more by his centrally integrated reading habits than by his perception time for individual words, so much so that he may be typed as being a slow reader or a fast reader, so will the motor habits set a psychophysical limit to performance on a new motor task. The fact that this limiting influence on performance may operate quite independently of the conscious effort of the individual should prove of interest to the educator, for example, who wishes to influence intellectual habits, or to the industrial specialist, who may wish to alter certain motor habits.

Indicator No. 16 is the memory component of the writer's time-sense test. It has a factor loading on C of .43. In this test the subject estimates a ten-second interval from memory by starting and stopping an electric clock without the aid of vision, after twenty and after thirty trials with the aid of vision of the clock face. His memory score is taken as the average deviation of his estimates from ten seconds, an average value usually achieved with the aid of vision. In this task we tap nonverbal aspects of the central integrative field with particular reference to its stability. Experience traces, built up during the trials with vision, play into C with more or less stability. Where their stability is high, they serve as accurate referents during critical recall without the aid of vision (i.e., when the perceptual component is removed).

Indicator No. 17 is the central form-discrimination component of the writer's dynamic visual field test. It has a factor loading of .41 on C. In this task the subject is required to record his discriminations of simple geometrical forms (square, circle, triangle) exposed for only twenty milliseconds at the foveae along with elements of the general task described as indicators Nos. 18 and 19. The minimal visual angle subtended by the figures is about 2° (viewed at thirteen inches), a value well above the threshold for form acuity. We have found that within the range of normal (Snellen) acuity, that is, from 0.8 to 1.2 vision, there is a zero correlation with performance on indicator No. 17. This fact is in line with our interpretation of the central integrative field as deriving in part from, but acquiring relative independence of, the sensory avenues. Only one of the three figures is exposed at a given time. Perceptual clues garnered during the brief exposure play into the central integrative field (much as the experience traces of our time-sense test No. 16), which is in a state of more or less phenomenal stability. Where the dynamic stability of C is high, its threshold to weak incoming communication from the sense avenues is correspondingly low with "accurate" objective performance given as a result. Its threshold is nevertheless very high in comparison with absolute limits of sensitivity (221).

Indicator No. 18 is the central color-discrimination component of the dynamic visual field test. This component is carried in the test by the same geometrical figures used for securing differential form discrimination. Thus, each of the three foveal figures is always seen as being red, blue, yellow, or neutral in color. (The targets are projected transparencies prepared with standard dyes and are not equated for brightness. We use the term "color" here to indicate differential pigmentation of the targets rather than to specify the technical basis of discrimination as being color vision per se.) It is of interest that the color dimension of the dynamic visual field test has an identical factor loading on C with the form dimension, namely, .41. As in the case of the form component, the critical threshold involved is probably that of the central integrative field, rather than the fovea itself or its associate primary projection pathway, and is considerably higher than the absolute threshold.

INDICATORS WITH LOW FACTOR LOADINGS ON C

While we have examined those of our indicators which have relatively high factor loadings on the central integrative factor C, our tentative interpretation of C must take into account those with low factor loadings. In other words, we must find a rational interpretation of C which provides

for its being substantially present in some of our indicators and substantially absent from others.

Indicator No. 1 is the Carl Hollow-Square Test. It has a marginal loading on C of .25. This is a standardized performance test for general intelligence, which yields an I.Q. It consists of twenty combinations of three or four irregular-shaped blocks. Each set, when properly assembled, yields a square pattern to be inserted in a square cutout. In addition to differences in shape, the individual wooden blocks have straight and beveled edges. The total time and the number of moves necessary for solution of each combination are recorded to yield a composite score. It is probable that performance on this test is influenced to a considerable extent by behavior which may be characterized as "vicarious trial-and-error behavior." While considerable abstraction is required in grouping according to related edges and shapes, the organized habits of the individual, C, contribute relatively little to the successful solution. The subject may actually try various arrangements in seeking a solution, in which case each move is counted and influences the final score. On the other hand, he may try several arrangements mentally or vicariously with only a single (successful) movement registering in his score. The total time required for solution probably inadequately reflects these vicarious trials. The solution for each of the twenty combinations is unique. Thus, there is little carry-over of successful or unsuccessful experience garnered from solution of the first combination to subsequent ones. As a consequence, no portion of the central integrative field becomes differentiated as the test progresses to contribute significantly to later successes. This is in marked contrast to our category test, indicator No. 2, for example, where a "success" principle is reinforced throughout by rewards and punishments.

Indicator No. 3 is the writer's test for critical-fusion frequency. It has a zero factor loading on C. In this test the subject adjusts the rate of flashing of an intermittent light source until fusion (appearance of steadiness of the light) occurs. He is given five preliminary trials in which to grasp the instructions and to reach a light-adapted state at test level following a dark adaptation period of five minutes. He is then given five critical trials which are averaged for a critical-fusion frequency value. There is no external criterion of success on this test, since the subject performs each trial without knowledge of his objective performance. The test is remarkably free from the influence of learning on subsequent trials. The subject performs at leisure and can reorient to the external stimulus at will when instabilities in the central integrative field might otherwise intrude upon objective performance. The test is nonverbal in character;

hence well-organized language patterns of the central integrative field cannot be mobilized to influence performance. We shall introduce experimental evidence in chapter ix which indicates that, while the essential factor reflected by this test is not of the nature of C, it is nevertheless related neuropsychologically in its functioning to C.

Indicator No. 4 is the speed component of the writer's tactual-performance test. Its loading on C is .12. The score employed consists of the total time for three trials to completion on the Seguin-Goddard performance test, presented at an angle of 70° with reference to the horizontal and without the aid of vision. The test objects consist of ten differently shaped wooden blocks which must be fitted into corresponding cutouts on the test board. At first impression, we might expect that the speed score of this test would reflect a kind of over-all mental efficiency and hence would show a relatively high loading on our central integrative factor. How can we account for the fact that its actual loading on C is zero? Here again we are faced with a test in which a background of well-organized habits (C) can contribute little to an early rapid solution simply because no such background exists. The subject, who normally employs visual clues in performing matching problems of the type presented here, encounters for the first time a situation requiring the use of tactual clues. A "new" and strange modality is called into play. Unless already totally blind, the subject reacts to being blindfolded with considerable feeling of frustration, sometimes almost catastrophic in intensity. He usually begins his matching on a trial-and-error basis, sometimes actually sliding the first block over the surface of the board until the appropriate recess is located (chance solution).

Not uncommonly the normal individual with even a high formal intelligence quotient will complete his first solution of the board without manifesting any signs of employing rational hypotheses or orderly methods of search. He may persist in his attempts to fit "square blocks into round holes" and vice versa to a degree sufficient to give the impression of stupidity to a casual observer. He is at the outset without ego control of the situation and reflects this fact by his disorganized behavior. Following the first three or four chance successes, he may or may not institute a more orderly method of search. He may begin to abstract significant clues afforded by the objects, such as straight edges, curved edges, presence and number of corners, absence of corners, number of edges, relative size of the objects, and positions in space. On the other hand, he may persist in his trial-and-error "gropings" until much later in the test and then only by sudden increase in his rate of progress give signs of abstracting. Following

the initial trial with the dominant hand, he is given a second trial in which he uses only the other hand. Once again the strangeness of the modality is uppermost, and he may react at first in a disorganized fashion. As the strangeness wears off, he turns once more to abstraction for successful solution. A third trial, given bimanually, usually requires the least time of all and, by its orderliness, reflects mounting ego control of the task, with receding anxiety. Speed, or total time for performance of the three trials, thus reflects the extent to which the individual is modality-bound in his approach to a psychologically "new" task.

Observations which we have made of two totally blind subjects, deprived of sight for several years, lead the writer to speculate that the speed component of this test would be found to have a substantial loading on C for groups of such subjects. It is hoped to secure adequate evidence on this point at a future date.

Indicator No. 5 is a memory component of the task presented for indicator No. 4. It, too, has a low factor loading on C of −.02. Upon completion of the third trial on the form board, and without any prior intimation to the subject, the board is removed from his presence, a blank sheet of white paper is placed before him, and he is asked to draw an outline of the board as he imagines it to be, including in their proper locations the shapes of the blocks. The score employed is the number of shapes out of ten correctly outlined. In this manner we test for the amount of incidental learning that has taken place during the three trials of the preceding portion of the test. This learning is too recent and too little reinforced to be incorporated as a differentiated part of the central integrative field. Its reproduction via diagram is not, strictly speaking, true memory, per se, for it has not yet had sufficient time in which to undergo the reorganizations and simplifications which are characteristic of true memory traces as they become organized portions of C. Rather, it is of the nature of immediate recall; and, in its resemblances to the original experience, it is more nearly like the eidetic imagery of children. Sharp corners are reproduced in the outlines as sharp corners, curved lines with accurate curvature; acute and obtuse angles are produced as such. In the accuracy of this recall is reflected the extent to which abstraction of significant elements has taken place in achieving successful performance of the initial tasks. We shall leave for a later chapter our consideration of the influence of a third factor which contributes to recall of the abstracted clues, namely, a power factor (P).

Indicator No. 6 is the localization component of the recall test referred to. Its loading on C is .19. The score employed for this test is the number

of correctly indicated shapes outlined in the diagram of the board which are correctly located as to relative position on the board. The experiential basis for successful performance on this indicator is very limited in comparison with that for recall of the blocks themselves. The clues for the distinctive shapes tend to be reinforced over a relatively large fraction of the total search time by punishment (in the form of increasing frustration) and again during the brief interval of successful matching by reward (in the form of lessening frustration). Reinforcement of the position clues is limited, by comparison, largely to the latter type. While some abstraction of significant position clues does occur, thus making possible some recall, the traces are less stable than those for the shapes of the blocks. They do not become well organized as part of the central integrative field, which fact is reflected by the vanishingly small loading on C. It is unfortunate, for our present purpose at least, that our battery did not include additional indicators specifically designed to tap imagery for spatial relations. It is quite possible that our localization component would be found to have a substantial relation to such ability. Since its experiential basis arises through the tactual modality, this indicator might prove useful in analyzing so-called "mechanical" abilities as encountered in certain analogous types of industrial situations.

Indicator No. 19 is the peripheral-vision component of the writer's dynamic visual field test (see discussion of indicators Nos. 17 and 18 for the central form and color components of this test). It has a low factor loading on C of −.15. In this test the subject must discriminate the presence or absence of a graduated circular patch of neutral light which appears somewhere in the peripheral visual field simultaneously with the exposure of a form and color target at the foveae (central vision). As may be seen from the intercorrelations given in Table 3, page 40, the form and color components presented together via central vision are highly correlated (.809), whereas peripheral vision, as tested here, has a zero correlation with form (.058) and color (.026), respectively. In addition to this evidence for the fact that we are dealing with a separate function in the case of peripheral vision, there is the now well-established fact (Halstead *et al.*, 206) that central vision in man (and other mammals) is projected to the striate cortex of the occipital lobes while peripheral vision is mediated by the cortex of the mesial surfaces of these structures. These functions are selectively disturbed by localized lesions of these respective areas of the cerebral cortex. We shall consider experimental findings in chapter xiii which indicate that these functions may also be selectively disturbed by accumulative effects of low-grade anoxia (of simulated high altitudes).

The negligible factor loading of peripheral vision on C with substantial loadings for central visual functions, although tested simultaneously, does not then necessarily represent an inconsistency in our findings. On the contrary, it is in line with expectancy; for, in marked contrast with central visual functions, peripheral vision ordinarily has little direct relationship to consciousness or awareness. Our recognition of forms or colors in the outer world is mediated directly by central vision. Peripheral portions of the visual field, coupled in mosaic fashion to motor-orienting reflexes in insects, contribute in man to the maintenance of fusion of our binocular vision and to the integration of vernier postural adjustments. In man, any portion of the peripheral field can be brought into consciousness (a fact which is of great importance to drivers of motor vehicles and to fighter pilots, for example), but this is not a habitual state. Our test situation, however, requires throughout that the total peripheral field be directly organized in the consciousness of the subject. This relatively unfamiliar situation gives rise to a condition similar to that produced by our tactual-performance test (indicator No. 4). Here it probably involves a realignment or redistribution of the habitual attention gradient associated with peripheral vision. During this process, modality considerations are prepotent during the early stages. Since our test affords but a single "practice" period, the performance score may be expected to reflect their influence primarily, rather than that of the central integrative field.

Influence of training on peripheral vision.—In a later chapter evidence is presented concerning the effects of experimental anoxia upon the peripheral vision component of the dynamic visual field test. As may be seen from examination of Figure 14, page 119, where these relations are dealt with quantitatively, repetition of this test on successive days by normal subjects without knowledge of their results yields a considerable degree of improvement in peripheral discriminative ability. This learning effect is so marked, in fact, that it becomes desirable to give practice until a plateau is reached before certain types of long-range studies are started involving serial measurements with the test. Such practice effects indeed represent an interesting type of learning which apparently occurs in the absence of a specified goal, in the traditional sense of the term, and in the absence of reinforcement in the sense of a so-called "law of effect," since the subject remains in ignorance of the objective accuracy of his responses. It is highly possible that this type of learning occurs in lower animals where our failure to grasp its significance may be contributing to the confusion in theories which surrounds that field at present (cf. 290, 365, 381, 404). We may speculate, in advance of experiments yet to be performed,

that the initial plateau referred to represents a point or stage in learning at which influence of the central integrative field gains temporary or permanent ascendancy over other factors in limiting further improvement of performance. In line with this hypothesis, we would expect the scores on our peripheral-vision test to show a significant factor loading on C at a stage where a performance plateau had been established through sufficient preliminary practice.

ESSENTIAL AND SUFFICIENT CONDITIONS FOR C

In arriving, in the foregoing discussions, at our interpretation of the C factor as the central integrative field factor, we have drawn our evidence from diverse sources. Our starting-point in each instance, however, has been the differential factor loadings of the various indicators. These loadings stand as objective summaries of certain facts concerning the population studied. Like all summaries, they do not tell the whole story. While they are themselves specifiable in operational terms, it does not follow that all the events for which they stand are necessarily so specifiable. In so far as the major variance on each of the indicators has been brought under laboratory control, or can be in subsequent studies, there i a genuine basis for hope that the events underlying the major variance can be described operationally. Factor analysis is primarily an exploratory tool. It is economizing in certain types and in some stages of behavioral investigations in that it provides rational bounds both for our search for related events and for our discourse (254). It does not define the factors isolated, although it may help to do so. Where, as in the present instance, we are concerned with defining behavioral factors in terms of basic functions of the organism, our critical evidence must come as an endless chain from laboratory studies. Each successive link is forged with the help of what has gone before. The reader will thus recognize that, since much critical evidence for specifying the essential and sufficient conditions for C, and for the other factors to be discussed in following chapters, is not yet available to us, our interpretations of these factors can at best be but tentative. With this fact clearly in mind, let us examine the remaining factors specified in the pattern of Table 4.

CHAPTER VIII

THE A FACTOR

IN THE battery of thirteen indicators represented in Table 4, page 41, it may be noted that four of the tests cluster together in column A in such a way as to signify the existence of a separate group or primary factor. These tests are as follows: Test 1 is the Carl Hollow-Square Performance Test for Intelligence; Test 2 is the Halstead Category Test; Test 5 is the Halstead Tactual-Performance Test (memory component); and Test 6 is the Halstead Tactual-Performance Test (localization component). We have described the nature of these tests along with the others in the preceding chapter. To avoid tedious repetition for the reader, we will limit our discussion here of our A factor to the four tests with substantial factor loadings for it.

In every instance those tests with low factor loadings on our A factor have a substantial loading (major variance) either on the C factor or on two others to be described.

Test 1 has a loading of .45 on our A factor; while No. 2 has a loading of .63; No. 5, a loading of .66; and No. 6, a loading of .34. The loadings for the remaining nine tests of the battery on our A factor are vanishingly small or zero in magnitude.

Test 1 is the Carl Hollow-Square Test. It is a performance (nonverbal) test for general intelligence which has been standardized on hospital populations. It consists of twenty combinations of three or four irregular-shaped wooden blocks, each set of which, when properly assembled, yields a four-and-five-eighths-inch-square pattern. Each block has straight and beveled edges which must be taken into account in assembling the combination. The score on this test is influenced both by the total time required for successful solution of each combination and by the total number of trial moves made toward assembling the blocks.

For each combination the subject is faced with the problem of comprehending the relatedness of marked differences in shapes and sizes, their critical positions in space, and their appropriate alignment of edges to yield the constant square pattern. From the marked variations in content presented, he must abstract unique groupings to a general criterion (square). To cast the statement in language which we employ elsewhere in

describing abstraction, he must grasp essential similarities in the presence of apparent differences and vice versa. He must accomplish this without very much to go on in the way of past experience. Not only is the general problem of the test an original one, but the solution for each combination is unique. Thus, even as the test progresses, he must still rely upon abstraction rather than upon recent learning for continued success.

FIG. 2.—Halstead category test (indicator No. 2). Subject sits before screen and views geometrical patterns exposed serially to him. The odd figure in each set indicates which one of four keys (in box on subject's table) should be pressed for correct response. Correct responses cause a soft chime to sound; incorrect responses, a rasping buzzer. Examiner sits to the rear, records responses of subject, and controls progress of test by keyboard resting on examiner's table.

Test 2 is designed to measure the ability of the subject to "abstract" various organizing principles, such as "size," "shape," "number," "position," "brightness," and "color," from a series of stimulus figures presented visually by means of a multiple-choice apparatus. A photograph of this apparatus is shown in Figure 2. The stimulus figures appear on the viewing screen in front of the subject, who registers his response by selecting one of the keys (electrical switches) on the panel of a response box

before him. The examiner, seated behind the subject, operates a control box which preselects the "correct" key on the subject's response panel. For each item, selection of the correct key by the subject automatically sounds a chime, while selection of any wrong key automatically sounds a buzzer, tuned to simulate the sound of a "raspberry" or "Bronx cheer." This arrangement of signals normally permits increasing certainty of principle for the subject as any given subtest progresses.

The test comprises 336 items which are divided into nine subtests. The first subtest consists of Roman numerals which correspond with illuminated numbers above each response key of the subject. This subtest establishes an association between the response keys of the subject and the stimulus figures of the viewing screen and thus "fixes" the verbal instructions for the test. It also permits rapid dissipation of anxiety associated with the apparatus.

Subtest 2 consists of 40 items in which the number of figures or groups of figures constitutes the organizing principle. In Subtests 3 and 4, oddity (significant difference in the presence of apparent similarity) is the organizing principle employed. Oddity is made effective through variations in such relata as solid versus broken lines, shape, color, brightness, and size. There is a certain progression in difficulty of items. Thus, through the latter part of Test 3 and the whole of Test 4, the perceptual prominence of the odd figure diminishes. This change is paralleled by an increasing demand for a logical-verbal approach to the test figures. This shift in emphasis is accomplished by an increase in the dimensionality of the test figures: If oddity is signified by the shape characteristic, differences in one other characteristic are introduced into the figures.

Subtests 5 and 6 involve a quadrant principle in space. The basic figure is an outline square divided into four quadrants. Views of this figure are presented as successive items, in each of which one of the four possible quadrants is missing. The missing quadrant in each instance is further accented perceptually by the fact that the quadrants given in the figure are numbered. These quadrants are arbitrarily numbered in a clockwise direction, beginning with I at the subject's upper left, as described on page 45. Quadrant II is his upper right, quadrant III is his lower right, and quadrant IV is his lower left. The quadrant principle is carried throughout these tests by a considerable variation in content. The first item in Test 5 presents three quadrants of a square. A Roman numeral appears in each quadrant, except for I, which is missing in the figure, and associates each quadrant with the numbered response keys. Tests 7 and 8 involve the part-whole principle as the basis for organization. Thus, key 1

is indicated for correct response if one-fourth of the figure, or of the numbered lines, is composed of solid lines, the remainder consisting of dotted lines. If one-half of the figure is in solid lines, key 2 is indicated; if three-fourths is solid, key 3 is indicated; and if the entire figure (of all the lines) is solid, key 4 is indicated as correct. Subtest 9 is composed of duplicates taken from Tests 2–8. It thus serves as a recognition test.

Several methods of scoring performances were considered. The one which has been adopted for quantitative comparison with other measures in this study is simply the number of errors.

There is a single principle of "oddity" or "difference" in each subtest. Following an initial period of trial-and-error groping on the first few items of each series, the organizing principle normally emerges or is "grasped" by the subject. The principle then holds throughout the remaining items of the particular series regardless of marked variations in their content. For example, where "differences in shape" is the effective principle, the "correct" figure in the item to be chosen by the subject may have any conceivable shape so long as its shape is unique with reference to the other figures in the item. For successful performance on the test the subject is thus forced to recognize recurrent similarities in the presence of marked dissimilarities and vice versa and to make appropriately differential responses. The ability to carry out such tasks has long been known in psychology as the ability for abstraction. Since this ability is a prime requisite for successful performance on Test 2, it would appear justifiable to identify it as a "factor of abstraction," or the A factor.

In what manner does a factor of abstraction, the A factor, enter into successful performance on the remaining two tests represented in the cluster of column A? These are the memory and localization components, respectively, of the writer's tactual-performance test. The basic test materials consist of a modified Seguin-Goddard form board with cutouts into which ten variously shaped wooden blocks of the same thickness are fitted by the subject. The subject performs this task three times while blindfolded. Without ever seeing the test materials, the subject is then asked to draw an outline of the board as he imagines it to be, including the cutout shapes and their approximate location on the board. Nothing in the initial instruction to the subject has suggested to him that he would be asked to make the drawing. He is thus forced to rely in part, at least, on incidental learning associated with three acts of performing the task without any visual clues. From tactual experience of straight and curved edges, square or rounded corners, acute, right, or obtuse angles, large and small blocks, he must develop a notion of the ten significant forms. Since

several of these properties are recurrent in different blocks, he thus must abstract appropriate combinations in order to be able to draw the outlines of the blocks. Similarly, from a changing set of tactual clues presented by the board in various stages of completion, certain constancies in space or location must be abstracted. This part of the task is the most subtle of all, since the opportunities for checking the relational distribution of the cutouts in space are limited largely to the brief intervals of critical choice.

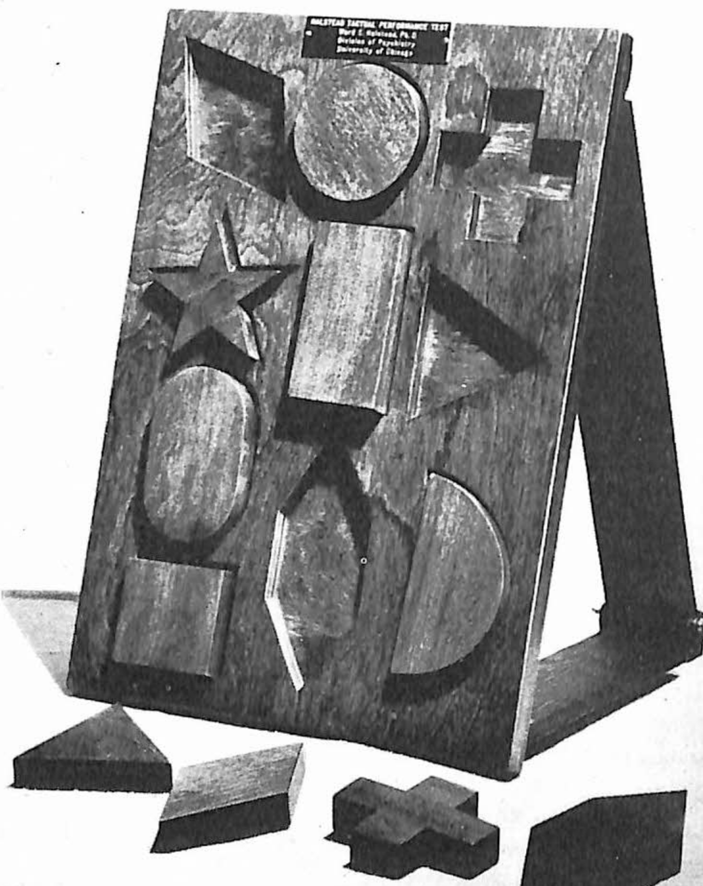


FIG. 3.—Halstead tactual-performance test (indicators Nos. 4, 5, and 6). Subject must insert wooden blocks into corresponding cutouts on tilted surface of a Seguin-Goddard form board while blindfolded. One trial for each hand and a final trial permitting use of both are given and timed. The total time for the three trials is the score for indicator No. 4. The subject subsequently makes an outline drawing of the board as he imagines it to be, indicating the various shapes (the number indicated correctly is the score for indicator No. 5) and their location on the board (the number located correctly is the score for indicator No. 6).

Its loading of .34 on A suggests that factors other than abstraction are important in this variable. Its total variance appears to be almost equally distributed among the four factors specified in Table 4, page 41. This fact may be of considerable significance to us in understanding learning behavior of which this indicator may be a prototype. Psychologically, the spatial ordering of experience is closely related indeed to the temporal ordering of experience regardless of the fact that mechanical considerations of the medium intervene to force a separation in communication. "Man is a time-binding organism," but it is no less true that he is also a space-binding organism. Accented time and space are the warp and woof

TABLE 5

DIFFERENTIAL IMPAIRMENT OF RECALL FOR OBJECT SHAPES AND THEIR LOCATIONS IN FRONTAL LOBECTOMIES, AS COMPARED WITH CONTROL GROUPS, ON THE HALSTEAD TACTUAL-PERFORMANCE TEST

Subjects	Indicators			
	No. 4 Speed	No. 5 Recall for Shapes (RS)	No. 6 Recall for Localization (RL)	Ratio RL/RS
Normal controls	9.5	8.4	6.1	.702
Military controls	12.0	7.8	5.6	.701
Miscellaneous controls	11.7	8.2	4.9	.600
Nonfrontal lobectomies	16.1	7.3	5.2	.701
Frontal lobectomies	21.7	4.9	2.5	.501

of our mental schema for the outer world. Yet, rarely do we attempt to accent them equally in our efforts to induce learning, whether in the laboratory or in the classroom.

There is a strong suggestion in our quantitative results that certain types of brain injuries may disturb the spatial-localization component established as incidental learning somewhat more than the recall for distinctive shapes similarly established. This fact is brought out in Table 5, the data for which are taken from the table given in Appendix D. Whereas our normal control groups and our nonfrontal lobectomies localize accurately about 70 per cent, on the average, of the blocks whose shapes are accurately recalled, our frontal lobectomies recall fewer shapes and localize accurately relatively fewer, only 50 per cent on the average. They also spend the most time on the average in performing the test of any group; i.e., they have the most objective experience with the content of the test.

The A factor and grouping behavior in brain-injured patients.—During the early exploratory phases of the present general investigation, it seemed desirable to secure information as to the ways in which relata of various kinds could be organized or integrated by normal individuals and by patients with cerebral injuries. For purposes of a general survey it was decided to utilize situations of the "open-field" or "projective" variety. Such situations differ from the more conventional "closed-field" type of test in that the task is defined only in general terms and the work unit of performance is bounded by the preference of the subject. Several investigators had previously claimed various degrees of success with such methods, particularly when supplemented by a clinical evaluation of the results (cf. 1, 106, 149, 384, 48, 162, 350, 425). In several instances, a relatively informal style employed by the investigator in reporting results made it extremely difficult to differentiate between descriptive and interpretative aspects of the work. The possibility that this is a general limitation to some extent inherent in the open-field method was recognized at the outset by the writer. Nevertheless, it was decided to establish as objective a procedure as seemed appropriate to the general problem and to the material available for study.

Several behavioral situations requiring the subject to group or sort heterogeneous test objects according to individual preference were devised. Among the various series of test objects employed with brain-injured individuals, with normal individuals, and with psychiatric patients were: a schematic face test similar to indicator No. 2, described in Appendix C; a color Gestalt test (indicator No. 25, Appendix C); a grouping test based upon playing cards; another based upon dominoes; still another based upon anagrams; a grouping test based upon abstract designs; and others based upon such materials as sticks of various lengths, an assortment of candies, an assortment of pictures, an assortment of tools, an assortment of colored yarns, an assortment of toys; a construction test based upon scrambled form boards; a sorting test involving tokens and onomatopoeic sounds; and a grouping test based upon sixty-two heterogeneous commonplace objects. While such materials proved very useful on occasion in demonstrating or pointing up certain anomalies in grouping behavior of individual patients with brain damage, the writer soon despaired of using them, with some exceptions, for isolating fundamental constants in behavior. To one interested in the possibilities of a science of psychopathology, this seemed reason enough to seek other methods. In justice to the open-field methods, however, it should be mentioned that they provide an unusually favorable opportunity for observing qualita-

tive aspects of behavior. As in the Rorschach situation (349), predominant personality trends such as "compulsiveness," "hysterical trends," "infantile attitudes," and the presence of "free-floating anxiety" are frequently focused for clinical evaluation by such methods. Hence, they are particularly useful in describing individual differences. Furthermore, it was from the background of experience with such methods that the battery of indicators used in the quantitative aspects of the present investigation was developed.

By making reasonable allowances for the clinical exigencies which arise, the open-field situations can be made to yield valuable information even at the quantitative level. It was from a study of this type that evidence was secured which led the writer to attempt the isolation of an A factor.

In that investigation twenty-six neurosurgical patients and eleven normal control individuals were studied. As in the case of the control group described in chapter v, the normal individuals were selected in such a manner as to secure maximum randomization of clusters of variables such as age, education, general intelligence, occupation, income, religious affiliation, etc. The neurosurgical patients are Cases 1–26 in the series described in chapter v. Among these, Cases 1–12 have a lesion in one frontal lobe, while Cases 13–26 have lesions in the brain posterior to the frontal lobes, including one instance (Case 26) of cerebellar lesion.

The test materials consisted of sixty-two objects which differed in such objective properties as size, shape, color, brightness, weight, material, hardness, and position. These were presented in a predetermined arrangement on the surface of a table. The subject was asked to "place those things together which seem to belong together." This instruction was repeated after successive groupings until the subject indicated that he could go no further. Following the first grouping of the materials, a recall test was instituted. The objects on the table were covered, and, after an interval of five minutes, the subject was asked to mention what was on the table. His replies were then tabulated as to whether the recalled objects had been grouped or had remained ungrouped. An "equivalence" test was then instituted wherein the examiner attempted to expand or contract each group prepared by the subject. In terminating the examination, simple categories of objects were constructed by the examiner, and the subject was then asked to guess the principle behind each category.

Examination of the groups made by the various subjects in terms of the equivalence test revealed the existence of four general types.

Type I.—This group was differentiated objectively by the fact that subtraction or removal of some of its content was rejected by the subject

as disturbing its organizing principle. For example, a group created by Subject M. H. consisted of a metal pulley, a glass stopper, a metal padlock, a picture of a house key, and a small metal key. The subject accepted subtraction from the group of the small metal key and the picture of the house key. He rejected subtraction of the metal pulley, the glass stopper, and the padlock. While the five objects were objectively equivalent in the sense of belonging to the same group for the subject, it became apparent that they were not equally dispensable to the group without disturbing its organizing principle.

Of the additions to the group offered by the examiner, the subject accepted (as not disturbing the organizing principle) another small metal key, an electric-light socket, a miniature light bulb, and a flashlight bulb. He rejected (as disturbing the organizing principle) a metal thimble, a metal spoon, some blue yarn, a piece of rubber-covered wire, a rubber grommet, a metal knife, a metal fork, a metal puzzle, a piece of coarse sandpaper, a wax crayon, a bottle labeled "Bergamot," a glass jar, a wooden pulley, and a picture of a bell. To summarize, certain additions or subtractions did not disturb the organizing principle, whereas others did. In examining the characteristics of the objects, such as size, shape, material, use, color, hardness, and weight, it was obvious that none of these provided the basis of organization for the group in question. The subject named the group "hardware."

Type II.—This group was characterized objectively by the fact that while all subtractions of content were accepted by the subject, all additions to the group were rejected. An example of this type is a group created by Case 25, consisting of a picture of a bell, a picture of a rooster, a picture of a rabbit, a picture of a doll, and a picture of a house key. Subtraction of any one of these objects from the group was accepted without disturbing the principle of the group for the patient. Among additions offered by the examiner, all of which were rejected, were a card labeled "Hairpin," a canceled foreign postage stamp, a card labeled "Pipe," a face-card playing card, a card with an abstract design (black dot and crescent), a piece of red paper stock, a ping-pong ball, a piece of coarse sandpaper, a smoked-glass lens, a piece of fine sandpaper, a metal puzzle, a metal knife, an electric-light socket, a pipestem, a wax crayon, a wax candle, and a piece of red cloth. The subtractions accepted by the patient indicate that the various objects were equally dispensable without disturbing the basis of the organization for the group. Since characteristics as to size, shape, color, material, weight, and use found in this group and in the additions rejected did not seem to be essentially different, they

apparently did not constitute the basis of organization of the group in question. The fact that no additions of test objects were accepted made it impossible to determine objective extensions of the equivalence range of the group without drawing upon material outside that provided in the test situation. The patient named the group in question "pictures." An examination of the additions to the group rejected by the patient shows that "pictures" for her did not include such objects as abstract designs, printing, and colored designs although these were objectively similar in size, shape, and material to objects in the group in question.

Type III.—This group differed objectively from the types thus far considered in that all subtractions of content were accepted and in that some of the additions to the group offered by the examiner were accepted, whereas others were rejected. An example of this type is a group created by Subject E. S. It consisted of a picture of a rooster, a black cube, a multicolored cube, and a square piece of red paper stock. Subtraction of each of these objects from the group was accepted by the subject without disturbing the principle of the group for him. Among the additions to the group offered by the examiner, the subject accepted a face-card playing card, a piece of coarse sandpaper, a card labeled "Pipe," a canceled foreign postage stamp, a picture of a bell, a small padlock, a miniature electric-light bulb, a glass stopper, a metal jar lid, a metal puzzle, and a wax crayon. He rejected some pink yarn, some blue yarn, a piece of red cloth, a glass jar, a round wooden box, a pipestem, a small metal key, a house key, a metal knife, a metal thimble, a small cork, a picture of a doll, a picture of a rabbit, and a card with an abstract design (black dot and crescent). The subject named the group in question "square things." Included among the additions accepted were such objectively "round" objects as the metal jar lid and the wax crayon. Among the additions rejected were objects similar in size, shape, and material to some of the original content. Groups of this type proved to be objectively undefinable upon examination of their equivalent and nonequivalent stimuli.

Type IV.—Groups of Type IV were differentiated from the foregoing types with respect to objective definability and the magnitude of the equivalence range. An example of this type was created by Case 10. It included a metal whistle, a knife, a metal pulley, a metal puzzle, and a house key. Subtraction of each of these objects from the group was accepted by the patient as not disturbing the organizing principle of the group for her. Of the additions to the group offered by the examiner, the patient accepted a metal fork, a small padlock, a miniature electric-light bulb, a lipstick, a metal spoon, a small metal key, a candleholder, a small metal chip,

a pipestem, some pink yarn with a metal tab attached, some yellow yarn with a metal tab attached, a metal jar lid, a flashlight bulb, some blue yarn with a metal tab attached, a doll slipper with a metal buckle, a piece of rubber-covered wire bared at each end, and a metal thimble. The patient rejected a card with an abstract design (black dot and crescent), a multicolored cube, a glass bottle labeled "Camphor," a glass stopper, a piece of red paper stock, a piece of fine sandpaper, a smoked-glass lens, a black wooden cube, a wooden pulley, a ping-pong ball, a picture of a bell, a glass jar, a round wooden box, a bakelite bracelet, a pipe bowl, a red poker chip, a bone chess pawn, a wax crayon, a piece of thick roundish stick, a rubber grommet, a piece of coarse sandpaper, and a picture of a

TABLE 6

SUMMARY OF AVERAGE GROUPING BEHAVIOR OF NORMAL INDIVIDUALS AND INDIVIDUALS WITH A CEREBRAL LESION

Subjects	Per Cent Objects Grouped	Total Per Cent Recalled	Per Cent Grouped Recalled	Per Cent Ungrouped Recalled	Groups Created				Grouping Principles Recognized
					Type I	Type II	Type III	Type IV	
Normals	80	67	77	23	16	5	1	5	5
Left frontal	27	26	26	26	10	1	4	0	4
Right frontal	41	34	34	32	12	2	4	1	4
Temporal	62	41	49	27	10	4	0	3	5
Parietal	90	61	71	22	18	4	1	4	5
Occipital	66	59	79	22	18	5	1	4	5
Cerebellar	87	66	72	25	24	5	1	4	5

house key. Upon examining the characteristics of the additions accepted and rejected, it became apparent that all the objects accepted as additions to the group were either part or all metal in material whereas no object rejected was in any part metal. The patient named the group in question "metal objects."

The relative frequency of these four types of groups as created by the various subjects in this investigation is shown in Table 6.

For every subject the groups most commonly produced "spontaneously" are those of Type I. The frontal-lobe cases are no exception in this respect. They are, however, differentiated from the other types of subjects both in the total number of groups created and in the relative frequency of Types III and IV. The frontal-lobe cases produced relatively more groups of Type III and relatively fewer groups of Type IV. From our description of Types I–III it is apparent that these groupings are characterized chiefly by irrational stability and involve organizing principles

not readily defined. They are, in other words, pseudo-categories. With reference to Type IV, the normal individuals produced on the average five such groups. Eight of the frontal-lobe cases, on the other hand, produced no groups of this type, while three cases (Cases 1, 4, and 9) each produced one such group, and one case (Case 10) produced two. In comparison with the normal individuals, the frontal-lobe cases produced a relatively large number of groups characterized by objective instability and a relatively small number of groups characterized by objective stability, i.e., pure categories.

The A factor and Type IV group.—At the beginning of the present chapter the A factor was identified as an ability for abstraction and defined as an ability to comprehend recurrent similarities in the presence of dissimilarities and vice versa. It is now apparent that it is just such ability that underlies the composing of Type IV groups. Groups of this type have three important characteristics:[1]

1. They represent pure categories in the sense that a single organizing principle is consistently applied to each of the objects or items of content.

2. Their size or range of content is infinitely extensible. Any object or event in nature which meets the criterion of the particular category may be admitted to the group.

3. Their size can be reduced until there is a minimum of two items or objects of content without disturbing the organizing principle.

In composing groups of this type, whether they take the form of pure number, music, natural science, painting, populations, elaborate filing systems, or reasoned argument, the A factor[2] is supported in varying degree by a third factor, which we consider next; namely, a power factor, or P.

[1] Results from a recent investigation, which we shall report in detail elsewhere, indicate that Type IV groups are further characterized by a redistribution or leveling of affective accents which renders the content *equi-available* in memory.

[2] For recent studies by other investigators which bear on our A factor see 73, 74, 75, 191, 193, 205, 210, 252, 253, 273, 290.

CHAPTER IX

THE POWER FACTOR P

PSYCHOLOGISTS have long recognized a class of behavior which is termed emotion or affective behavior. According to McDougall (301), for example: "Our primary emotions are rooted in our instinctive dispositions, and that instinctive striving and emotional expression are but two inseparable aspects of one activity. . . . . Affect denotes the emotional-conative aspect of all mental activity, with the recognition that feelings of pleasure and of pain are conditioned by, and in turn react upon, the cause of such activity, furthering or checking it, respectively, in proportion to their intensity" (p. 71).

The affects may be so vividly in the ascendancy in a given behavioral situation as to give rise to temporary general inhibition of thought, a state which Ferenczi, for example, has characterized as "affective feeble-mindedness." Anxiety may so overwhelm the thinking capacity of an individual as to mask it entirely. Clinically depressed individuals not uncommonly complain that they cannot think, owing to the intensity of their feelings. As their anxiety recedes and the feeling intensities diminish, their thinking capacity may be restored. (Our experience suggests that where the pathological process is long sustained, it may lose its character of apparent complete reversibility.)

Motor concomitants of affect and brain localization.—Electrical-stimulation and extirpation studies of the orbital surfaces of the prefrontal lobes in cat (373) and monkey (9, 10, 13, 353, 373) have revealed evidence that suggests some degree of specialization of function of these areas for such motor concomitants of affect as changes in respiration, blood pressure, and peristalsis. It is in this region of the brain that Kleist (268) localized "moral insanity." Bailey and Bremer (9) found that electrical stimulation of the proximal end of the cut vagus nerve in cat reveals a localized area of electrical facilitation in the orbital surfaces of the prefrontal lobes (area 13). This is of particular interest in view of the fact that Dragstedt (113) has found it possible to relieve gastric-ulcer pain in man by sectioning the vagi.

However, the most striking evidence for localized control of motor concomitants of affect has accumulated from electrical-stimulation and extir-

pation studies of particular portions of the hypothalamus. It has been observed (183) that transsphenoidal stimulation by means of an electrode pushed into the nasal surface of the sphenoid bone, at about the middle of the floor of the sella turcica, produced acceleration of respiratory and heart rate, dilation of the pupils, elevation of blood pressure, increase in vasomotor tone, rise in body temperature, sweating, contraction of the bladder, subjective feelings of marked anxiety, and uncontrollable sobbing. While the localization is by no means precise with this method, the results are, in general, in line with clinical and pathological findings of others and with studies carried out on infrahuman forms.

Wheatley (429) has made a careful study of changes in temperament of cats subjected to bilateral lesions placed in the hypothalamus with the aid of the Horsley-Clarke stereotaxic instrument. He found that lesions destroying the region of the nucleus hypothalamicus ventromedialis were invariably associated with savage behavior of an extreme type, with complete loss of any friendly attitude, although the animals had initially been selected for unusual tameness and friendliness. Lesions placed elsewhere failed to produce any comparable change.

It appears that convincing evidence is now available, from such studies as the foregoing, which supports a conception of affective behavior distinct from cognitive processes. Furthermore, implicit in this evidence is the notion of a dynamic rivalry or competition between affect-controlled behavior, on the one hand, and mentation-controlled or rational behavior, on the other.

We may agree with McDougall that affect, within normal or appropriate limits, operates as a kind of mordant for experience which helps bind together the saliencies of our central integrative field factor C. Nor is it difficult to concede that some ultimate "pleasure-pain" principle operates in the fixing of new ego boundaries, although a universal law of effect has not yet been satisfactorily documented in the laboratory. The psychologically "old" (C) not only has been tried but has been found pleasurable or painful. Our difficulties arise when we attempt to account for the essential continuity of ego growth-differentiation, for the ego does not appear full blown at any stage in the life-history of the individual. How does our A factor (abstraction), the vehicle of healthy ego growth, gain ascendancy over affective demands which are immediate as regards time and space? Must we not seek out a third factor which serves to buffer these affect demands in the face of the psychologically "new," holding them in abeyance until the A factor can make its penetrations into the entropies of unstructured time and space? For most certainly it will prove to be a factor which

has been strengthened from species to species, until it now underlies man's superior capacity for ego growth. Let us turn once more to our factor pattern in Table 4, page 41, for laboratory evidence of the existence of this third factor in biological intelligence.

From the entries in column P of this table it is apparent that there is another factor to be considered which is neither C nor A. Its representations from the total battery are drawn from four indicators: No. 3, the writer's flicker-fusion test; No. 5, the memory component of the tactual-performance test; and Nos. 17 and 18, the central form and color components, respectively, of the writer's dynamic visual field test. The most substantial representations for this factor are for indicators Nos. 3, 17, and 18. What is it that these variables have in common that is different from other variables in the battery?

In the case of indicator No. 3 it has been possible to simulate the test conditions for one of the above variables and to apply them to normal and to brain-operated monkeys at the same time that records of electrical activity of the brain of the animal were being obtained. In this way the writer and his associates have been able to demonstrate a direct relationship between brain-wave activity and certain essential features of the flicker-fusion test, indicator No. 3 (203, 204, 417, 418, 433). The flicker-fusion test requires the subject to adjust at leisure the flash rate of an intermittent source of light until the light appears to him as steady. The frequency of flashes at which this effect appears is known as the critical-fusion frequency (c.f.f.). We have found it possible to "drive" the brain waves of the monkey in one-to-one fashion up to frequencies corresponding to critical-fusion frequency by means of intermittent photic stimulation. A similar driving effect has been described in normal man by Adrian and Matthews (5), by Toman (405), by Durup and Fessard (115), by Loomis, Harvey, and Hobart (297), and by Jasper (247), who noted a correspondence between maximal driving frequency and the critical-fusion frequency in two subjects. Wang (421) demonstrated that repetitive photic stimulation of the retina induced electrical responses of corresponding frequencies in the visual cortex of the cat. Similarly, both Bartley (14) and Bishop (39) found that cortical electrical activity of the rabbit could be markedly altered in amplitude and rate by intermittent photic stimuli. Maximal effects were obtained at approximately five flashes per second, which frequency they regarded as the alpha frequency for the rabbit. Over a considerable range of frequencies, they were able to raise or lower the alpha rate by changing the rate of retinal stimulation. In the cat, Bishop and O'Leary (40) found the responses in the cortex from electrical

stimulation over a wide range of frequencies. In contrast, the electrical responses to photic stimulation were less predictable.

In the monkey we have found that the electrical activity of visual structures below the cortex (i.e., the optico-geniculo-striate system) could be driven to frequencies above critical-fusion frequency. Brecher's (51) results indicate that for the range of intensities employed by us, fusion

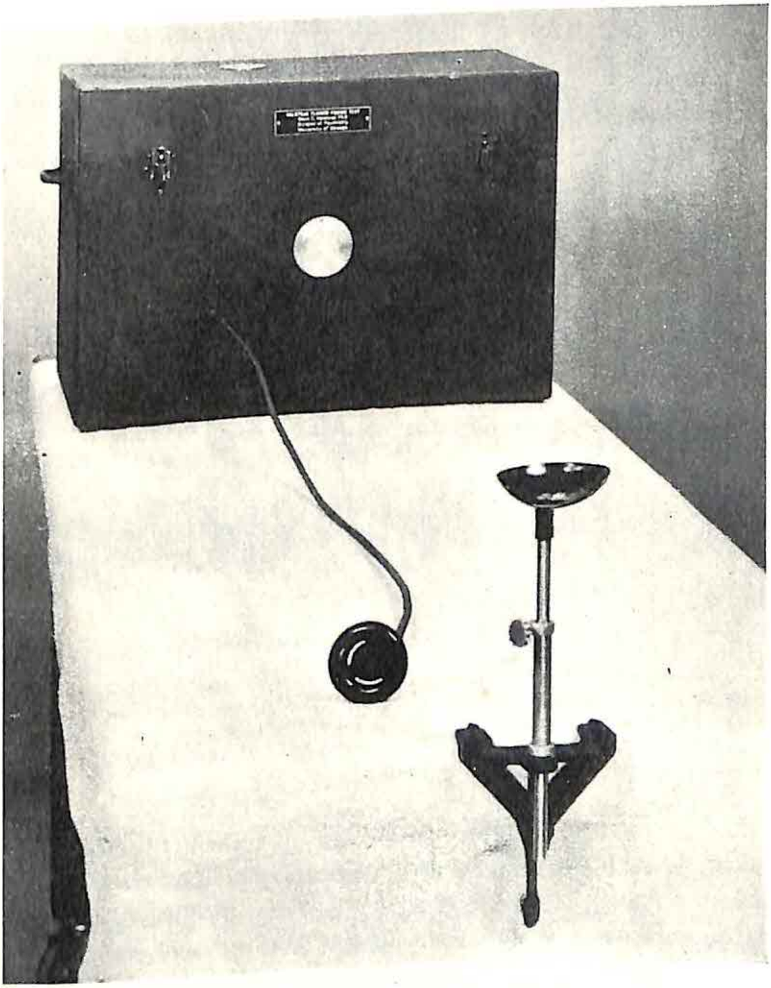


FIG. 4.—Halstead flicker-fusion test (indicators Nos. 3 and 3*a*). Subject sits with chin in chinrest in darkened room and views binocularly a flashing light in circular window of box. By means of control knob on flexible shaft, he adjusts flash rate of light until it appears to be steady. Examiner reads adjustment by subject in cycles or flashes per second from dial in top of box. Five preliminary trials are given to establish instructions and proper level of light adaptation. Five additional trials are then given, and their average result is taken as the score for indicator No. 3. Their average deviation is the score for indicator No. 3*a*.

occurs for the monkey at several cycles per second below that for man. Our observations thus eliminate the retina (the mechanisms of which have been regarded traditionally as providing the basis for temporal fusion) as the limiting factor on the temporal resolving power of the visual system. Taken with the above-mentioned lines of evidence, they indicate the presence of a fusion mechanism in the brain, more specifically, in the cortex of the brain. It seems very probable that the higher the rate at which the brain waves of an individual can be organized or reactivated

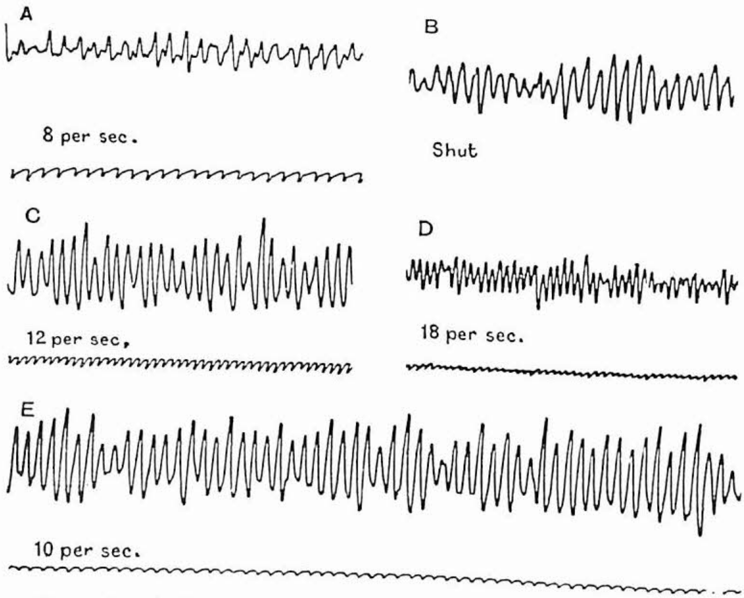


FIG. 5.—Examples of different brain rhythms induced in man by flickering light. Signal line shows frequency of flicker. *A*, flicker at 8 per second; *B*, eyes closed and field dark, Berger rhythm at 10 per second; *C*, flicker at 12 per second; *D*, flicker at 18 per second; *E*, flicker at 10 per second (compare with *B*). (Reproduced from E. D. Adrian and B. H. C. Matthews [5] with permission of the publishers of Brain.)

from the periphery by an intermittent light of a given intensity, the higher will be the critical-fusion frequency of that light for the individual.

Fusion on the subjective side is paralleled by decoupling or "escape" to endogenous rhythms by the brain waves. Without such an escape mechanism the complete transposition of behavior from daylight illumination to artificial illumination by sixty-cycle (suprafusional) current would not be possible. In the case of critical-fusion frequency, the point at which escape or decoupling occurs for a given intensity of intermittent light is determined by the relative magnitude of competing stimuli from other sources, either endogenous (intracerebral) or exogenous (from the periphery).

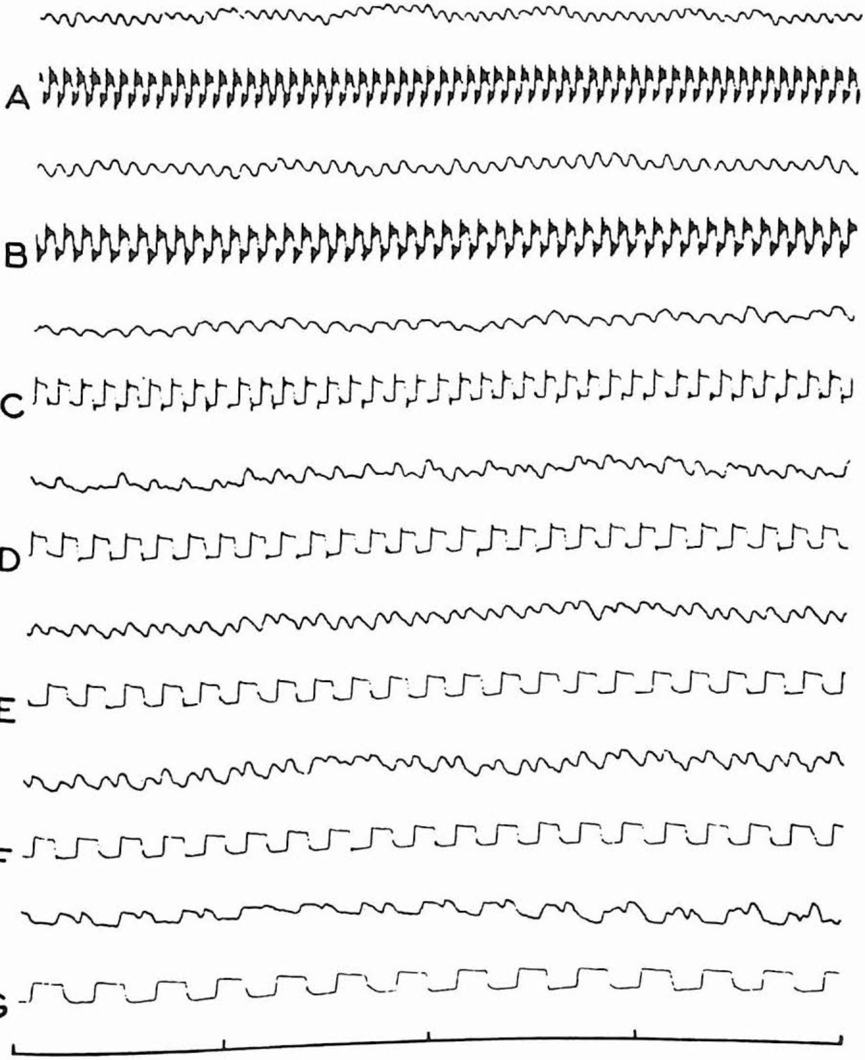


FIG. 6.—Alteration of brain rhythm of a monkey by flickering light. Signal line opposite each letter indicates frequency of flicker. Time line in seconds at bottom. *A*, flicker at 15 per second; *B*, flicker at 11.5 per second; *C*, flicker at 9.5 per second; *D*, flicker at 7 per second; *E*, flicker at 5.5 per second; *F*, flicker at 5 per second; *G*, flicker at 3.5 per second. Note tendency of brain rhythm to double flicker frequency in *E* and *F*.

Support for this interpretation is provided by our experimental observations that, when such competing stimuli are eliminated or reduced in value by low-grade barbiturate anesthesia, the range of photic driving is extended (of interest in connection with mental-status examinations of patients in barbiturate interviews [cf. 42]). Also there is the fact that an orderly (logarithmic) relationship is known to exist between critical-fusion frequency and the intensity of the intermittent light (222). In other words, once decoupling (fusion) has occurred, it can be restored immediately by increasing the intensity of the driving source.

In the case of the writer's dynamic visual field test, competing stimuli are systematically imposed on the perceptual field by requiring foveal form and color discriminations at the same time that peripheral-contrast discriminations are being made. The various stimuli are presented simultaneously in the perceptual field for an interval of only twenty milliseconds. The temporal resolving power of the brain for multidimensional stimulation of fixed intensity, as in the case of critical-fusion frequency, is thus limited by the intrusion of extraneous (uncontrolled) endogenous and exogenous stimuli. It is of interest that mild or severe degrees of altitude anoxia alter the dynamic visual field in a systematic fashion. The dynamic visual field is also altered by brain lesions in man. It is possible that the changes in peripheral vision found by Kennard (255) and others in the macaque monkey following unilateral or bilateral localized lesions in the frontal lobes likewise represent alterations in the dynamic visual field.

Enhancement of brightness below fusion.—To turn once again to the relation of intermittent photic stimulation to brain mechanisms, there is another line of evidence which supports the idea of a dynamic coupling between the periphery and the cortex. Bartley (18) has carefully explored the sensory resultant of intermittent light viewed at frequencies throughout the range below fusion. In this region the most striking perceptual feature is flicker. Since the total luminous flux of regularly intermittent light is independent of flash frequency, it has been rather generally assumed that apparent brightness within the range of flicker could likewise be expressed directly as a function of the product of the luminous flux and the time, i.e., independently of flash frequency. This has not proved to be the case. Using serial flashes of light, Bartley has found that very soon after flash frequency falls below critical-fusion frequency the apparent brightness of the intermittent field begins to rise until it reaches, then exceeds, a level equivalent to that obtained with steady illumination. The maximum enhancement is reached at eight to ten flashes per second. Noting the coincidence between the rate necessary for maximal enhancement

and the alpha rhythm commonly obtained in the human electroencephalogram, Bartley concluded that a "resonant" type of coupling occurs between the two trains of events with enhancement of apparent brightness as a resultant.

Muscular origin of enhancement eliminated.—Halstead (192) suggested that one effect of intermittent photic stimulation could be to "drive" the pupillary and accommodative mechanisms of the eye. Their failure to "follow" flash rates might result in opening the eye to more light or in changing the extent of light distribution on the retina, thereby enhancing apparent brightness. The writer investigated this possibility in a normal subject by eliminating both the pupillary and accommodative reflexes by scopolamine. Brightness enhancement was clearly retained in the absence of both reflexes. Thus the enhancement effect could not have been due to either of the intraocular reflexes. Halstead and associates (203, 204, 418) subsequently found a similar absence of any effect of mydriasis on the driving of cortical activity in the monkey by intermittent photic stimulation.

Critical-fusion frequency and brain injuries in man.—There have been no previous controlled studies on the effects of brain lesions in man on critical-fusion frequency for intermittent projected light. Werner and Thuma (427) have compared birth- (brain-) injured children with children who made low scores on standardized tests for intelligence and found c.f.f. to be reliably lower on the average in the former. They noted that the difference was most marked at low-intensity levels for the intermittent light and virtually disappeared for maximal c.f.f.'s. Werner and Thuma failed to mention the auditory component in their mechanical apparatus, however, and it remains unclear to what extent this may have been an uncontrolled variable in their results. However, Halstead (193) found a similar effect of low level of intensity in a patient with left prefrontal lobectomy in comparison with normal control subjects where the auditory component was controlled by elimination. This finding led to the adoption of a relatively low level of light intensity as the fusion task for our indicator No. 3. The apparatus which we developed for this task, after considerable preliminary study of mechanical types, is electronic in type. Flashes of constant intensity and duration (ten microseconds) are obtained from a cold cathode neon lamp, driven with a regulated thyratron circuit. Lamp intensity is standardized in electrical units by means of a built-in photometer; flash frequency is calibrated absolutely against a built-in sixty-cycle synchronously vibrating reed; and the auditory component is eliminated by housing the apparatus in an acoustically treated

chamber. The subject sets the light frequency at his fusion point by adjusting a single control knob. His setting is then read directly, in cycles per second from a built-in calibrated scale, by the examiner. The subject is kept in ignorance of his settings throughout. The range of light frequencies obtainable from the apparatus is continuous from ten to sixty-two cycles per second. As may be seen from the data given in Tables 7 and 8 and in Appendix D, this range is about 100 per cent greater than necessary to cover the c.f.f.'s of our experimental subjects. Since this range is available to the subject, it enables us to analyze his variance from trial to trial for sharpness of cutoff point as a clue, among others, to degree of co-operation of the subject.

TABLE 7

AVERAGE CRITICAL-FUSION FREQUENCIES IN CYCLES PER SECOND AND AVERAGE DEVIATION ON FIVE TRIALS IN VARIOUS EXPERIMENTAL GROUPS OF SUBJECTS

SUBJECTS	INDICATORS	
	No 3 Flicker Fusion cps	No. 3a Average Deviation cps
Normal controls	23.2	1.2
Military controls	24.8	1.1
Miscellaneous controls	18.6	.8
Nonfrontal lobectomies	21.7	1.2
Frontal lobectomies	17.2	.5

In Table 7 are presented the average critical-fusion frequency values in cycles per second and the average deviation on five trials for the various groups of subjects of the present investigation. These groups are as follows: normal control subjects, military subjects (controls), miscellaneous controls (including psychiatric patients), nonfrontal lobectomies, and frontal lobectomies.

It may be noted that in comparison with the normal and military controls, there is a tendency for our nonfrontal lobectomies to fuse at somewhat lower values ($P < .056$). This tendency is even more marked for the frontal lobectomies ($P < .001$).

Of no less interest and significance, however, is the fact that the average deviation of the frontal lobectomies is reliably less than the combined mean deviation for the normal controls, military controls, and nonfrontal lobectomies ($P < .001$). The data for individual patients are given in Table 8. This finding means that the frontal lobectomies are objectively

more accurate (less variable) in locating their fusion point on successive trials than the normal individuals or the nonfrontal lobectomies, although they fuse at relatively lower values in comparison with either.

These findings provide us with important clues as to the nature of the processes reflected by critical-fusion frequency. Here, for the first time,

TABLE 8

AVERAGE CRITICAL-FUSION FREQUENCY IN CYCLES PER SECOND (NO. 3) AND AVERAGE DEVIATION ON FIVE TRIALS (NO. 3a) FOR INDIVIDUAL SUBJECTS ACCORDING TO TYPE OF BRAIN INJURY*

LOBECTOMIES

(1) Frontal		(2) Temporal		(3) Parietal		(4) Occipital		(5) Cerebellar	
No. 3	No. 3a	No. 3	No. 3a	No. 3	No. 3a	No. 3	No. 3a	No. 3	No. 3a
17.2	.3	23.4	1.6	12.6	.2	24.3	1.0	28.4	1.6
18.6	.4	26.0	1.3	28.3	1.6	27.5	1.4		
19.2	.3	17.2	1.4	26.2	1.5	23.2	1.3		
17.6	.2	12.6	1.8	19.1	1.2	17.3	1.0		
20.1	.3	24.3	1.1	23.0	1.2	24.2	1.8		
15.8	.4	27.6	.8	16.0	1.2				
14.4	.5	24.2	1.2						
16.7	.5	10.9	.6						
22.6	1.1	18.5	.4						
21.2	1.3								
18.2	.3								
17.5	1.1								
17.4	.9								
14.8	.2								
14.2	.8								
15.1	.3								
17.3	.4								
13.8	.3								
20.1	.6								
16.7	.5								
18.3	.3								
16.7	.4								
17.2	.4								
17.2	.3								
11.7	.6								
Av. 17.2	.5	20.5	1.1	20.9	1.2	23.3	1.3	28.4	1.6

* For data on normal subjects compare Table 15, Appen. D.

we have direct evidence that they are central (cerebral) processes rather than peripheral (retinal) as they have traditionally been regarded. In view of our evidence for a coupling effect between flash frequency and brain-wave frequency in the monkey (203, 204, 417, 418, 433), it seems very probable that flash frequency of our indicator No. 3 is loading the brain-wave system in varying degree. Coupling is direct up to the point (frequency) where fusion occurs, at which point escape or decoupling occurs.

It is a familiar fact that both electronic and mechanical systems may fail or distort more or less acutely as a function of a power factor. This is notably true of sound-amplifying systems, for example, where the ability of the system to handle an input signal of, say, one thousand cycles without distortion provides one way of estimating the undistorted power factor of the system. A more commonplace example is afforded by the problem of estimating the usable horse-power of two automobiles. They may be undifferentiated in their performance at a speed of thirty miles per hour. They become markedly differentiated, however, as they are put acutely under critical load. The one with inadequate power reserve will fail acutely within a narrow range of variance, whereas the other, with good power reserve, fails only gradually over a somewhat wider range of variance due to the operation of factors other than power. The analogy here is with our frontal lobectomies, who, having a low reserve of power factor (reflected by low c.f.f.), fail to resolve flicker below fusion at an earlier point under loading from the test than the normal; but failure occurs within a narrower range of variance than the normal because of an inadequate power factor.

Cyclic variation in critical-fusion frequency.—In connection with our analysis of the central integrative field factor C, it was noted that flicker fusion, indicator No. 3, has a zero factor loading on C. This is in line with the fact that the indicator is normally remarkably free from influence by learning. There is reason to believe that where investigators have obtained unstable results for c.f.f., they have too readily overlooked instrumental sources of variance and too readily invoked "lack of co-operation" or learning as explanations.

We have found that c.f.f. and its average deviation may vary considerably throughout the day in the normal individual. To what extent it fluctuates with blood-sugar level or with other biochemical factors we have not yet had an opportunity to determine. That c.f.f. does fluctuate in a daily cycle is well demonstrated in the curves of serial determinations made over a period of weeks on the same individuals, as shown in Figure 7.

Of equal interest is the evidence in the curves of a much slower cyclic variation over a period of weeks. Two of the subjects are females. The slow cycle is independent of the menstrual cycle, however. Furthermore, it is almost as marked for the male subject. It will be of interest to explore these relations further with particular reference to thyroid and other endocrine functions.

Photic driving and brain lesions in monkeys.—Halstead and his associates have found that whereas the dominant brain-wave rhythm in the monkey electroencephalogram can be driven up to the point of critical-

fusion frequency only, the visual pathway below the cortex can be activated by photic stimuli to the retinas at suprafusional values (417). This fact is in line with our evidence above, which links critical-fusion frequency with cerebral processes, and especially frontal-lobe processes, in man.

Woolf, Walker, Knox, and Halstead (433) have explored the effects of lesions of the visual system upon photic driving in monkeys. Eight mature monkeys (*Macaca mulatta*) were studied in the following preparations:

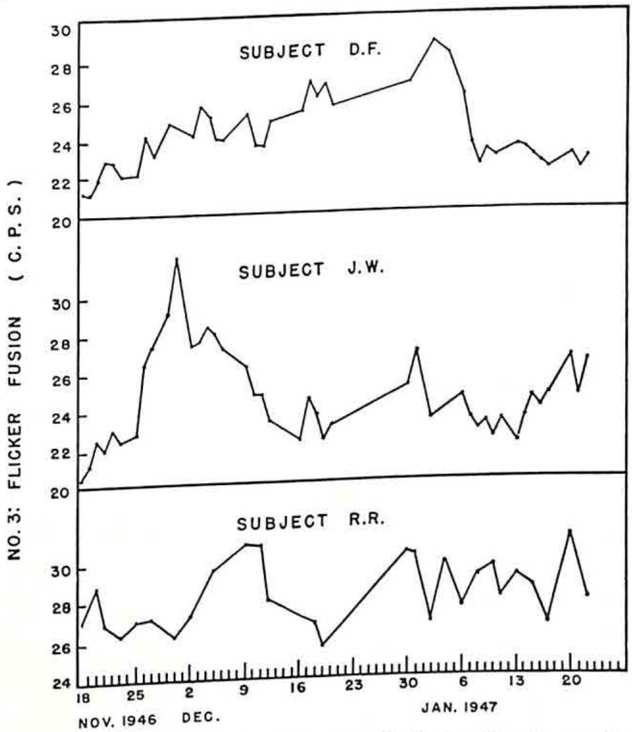


FIG. 7.—Fluctuations in flicker-fusion frequency (indicator No. 3) over days, weeks, and months in three normal subjects, ages twenty-two to twenty-four. Peaks seem to be related to "work efficiency." Each point on curve is average of twenty determinations.

1. Cauterization of the macular area of the retinas (two monkeys).
2. Partial (superior) sections of both optic tracts after anterior temporal lobectomy (two monkeys).
3. Bilateral anterior temporal lobectomy (one monkey).
4. Bilateral ablation of the occipital operculum (two monkeys).
5. Bilateral removal of the lingual gyrus (one monkey).

Lesions involving the retinas or the optic tracts were found to reduce the percentage of photic driving of the electroencephalogram in comparison with preoperative values. (Bilateral anterior temporal lobectomy, on

the other hand, seemed to produce an opposite effect.) Lesions of the striate cortex likewise reduced the percentage of postoperative photic driving. This was true whether the lesion involved removal of both occipital opercula on the dorsal surface or of both lingual gyri on the ventral surface. That the reduction in photic driving was marked in the cases of the opercular lesions (Monkey V) is shown in Table 9, where pre- and postoperative percentages of photic driving of the electroencephalogram

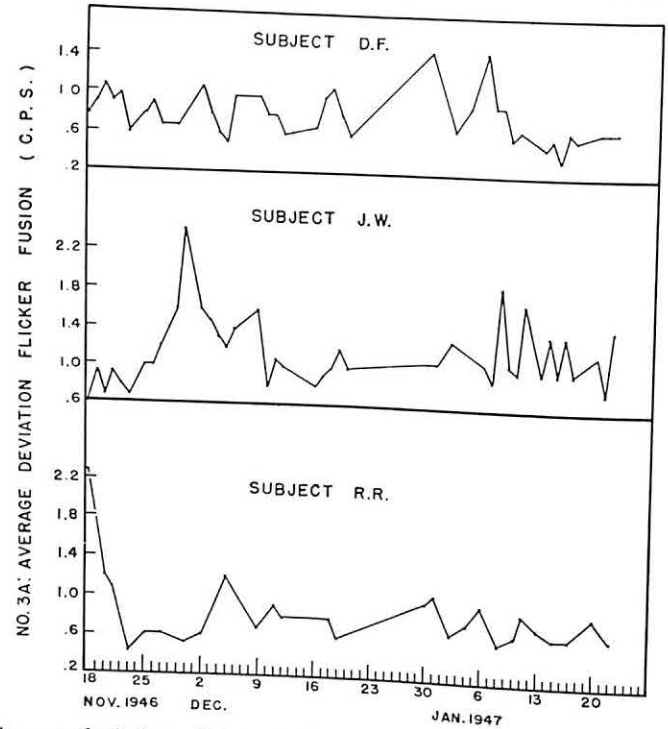


FIG. 8.—Average deviations of flicker-fusion frequency (indicator No. 3a) over days, weeks, and months in three normal subjects represented in Fig. 7.

through the frequency range from one to sixteen cycles per second (light-dark ratio, 1:1) for a fifteen-second sample at each frequency are given. Following preoperative tests, both occipital opercula were ablated in Monkey V by Dr. A. Earl Walker on July 24, 1942. The first postoperative tests were made twelve days later.

This animal was sacrificed on October 1, 1942. Examination of the nervous system revealed that the lesions had involved the greater portion of the dorsal surface of the occipital lobes. Microscopically, the degeneration in the lateral geniculate bodies was found to be symmetrical for both sides. The anterior portions of each revealed almost complete degeneration

in a median sector. The lateral and medioventral portions appeared to be quite normal. At the posterior pole in the right lateral geniculate body only a few normal cells remained in layer 6 while the other layers were almost completely degenerated. At the posterior pole of the left lateral geniculate body a wedge-shaped area of degeneration was present with normal-appearing cells in the lateral and medial angles.

These observations, along with our evidence for reduction of c.f.f. in frontal-lobe lesions, are of interest in connection with another type of visually induced behavior.

TABLE 9*

PHOTIC-DRIVING PERFORMANCE OF MONKEY V BEFORE AND AFTER BOTH OCCIPITAL OPERCULA WERE ABLATED
(In Percentage)

Intensity in Foot-Candles	Filter Wratten No.	(7/21/42) Preop.		(8/5/42) 1st Postop.		(9/3/42) 2d Postop.		(9/14/42) 3d Postop.	
		R.†	L.‡	R.	L.	R.	L.	R.	L.
4	Blue (75)	40	65	15	20	10	0	0	0
4	Red (70)	25	35	0	0	15	5	0	0
4	Neutral	30	35	5	10	0	0	0	0
40	....	55	75	10	25	0	0	0	0
80	....	55	50	0	10	5	0	0	0
120	....	65	70	0	0	5	0	0	0

* After Woolf, Walker, Knox, and Halstead (433).

† Right hemisphere of brain.

‡ Left hemisphere of brain.

Critical-fusion frequency and optokinetic reactions.—It is now well established that optic-pursuit nystagmus (head or eye) can be elicited by moving vertical or horizontal striations across the field of vision at an appropriate rate in man and in many infrahuman forms, including invertebrates (189, 359, 360, 367, 369–72). The striations may take the form of black, parallel, equally spaced vertical stripes on a solid white background (or vice versa) which is the inner surface of a drum or cylinder which may be rotated around the subject. A photo-diagram of such an apparatus, which we have used in studying optokinetic reactions of human brain-injured and normal subjects, is shown in Figure 9. The subject, in this instance a patient with both prefrontal lobes removed, sits inside the motor-driven rotating drum. The drum is internally illuminated by a D.C. lamp. As the black stripes on the inner surface of the drum move through the visual field of the subject, he tends to fixate each one and to follow its movement out to the periphery by a horizontal eye movement.

(Head movements are eliminated by a head clamp.) Fixation of the stripe is broken in the periphery, and the eyes sweep back to center in a saccadic (rapid) movement where fixation occurs for another stripe and the cycle is repeated. The nystagmic eye movements may be recorded on paper tape through electrical pickup of variations in the polarity potential of the eyes by the apparatus shown on the left side of Figure 9 (cf. the writer's description of this method of recording eye movements in man [189]).

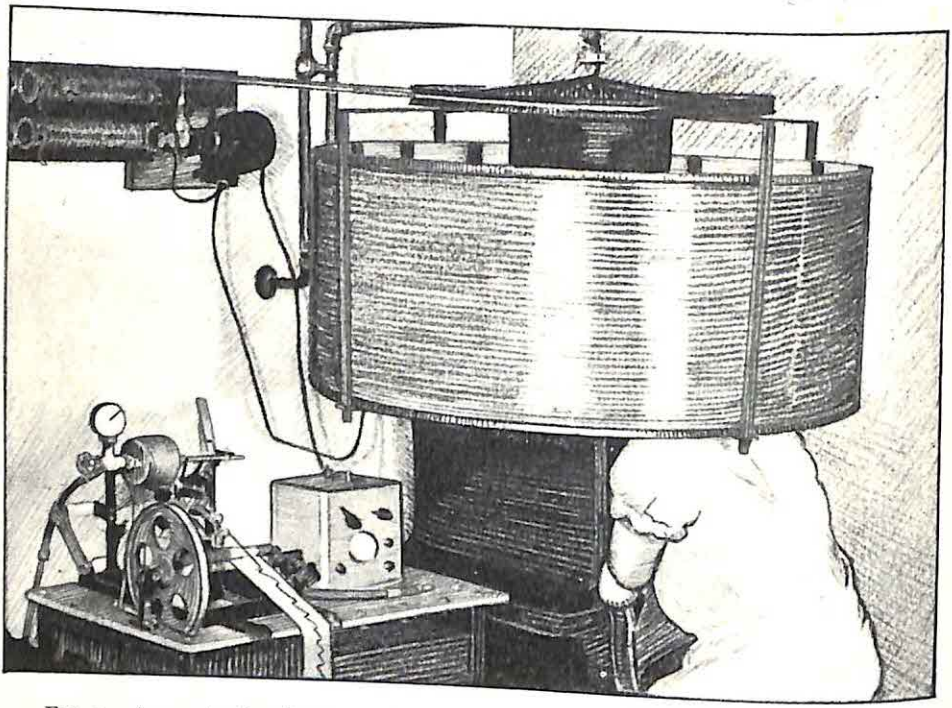


FIG. 9.—Apparatus for eliciting and recording optokinetic responses. As drum with striped pattern on inner surface rotates about eyes of subjects, reflex eye movements are elicited which are recorded on paper tape through electrical amplification of the corneo-retinal potential of subject's eyes.

Several investigators have reported that optokinetic reactions tend to be deficient or lacking when the movement of the stripes is toward the blind portions of the two eyes in individuals with homonymous hemianopia (146, 147, 389). Other investigators, including the writer, have found these alterations to be temporary following unilateral removal of either the right or the left occipital lobe (88, 165). The writer has been unable to demonstrate any reliable alterations in optokinetic reactions for vertical stimuli following neurosurgical removal of either occipital, parietal, temporal, or prefrontal lobes unilaterally or the prefrontal lobes bi-

laterally, either as to frequency or as to amplitude, in either right or left directions of rotation of the stripes, in carefully selected subjects. These observations appear to be in line with those of Ter Braak (49) for various infrahuman vertebrates. He found no reliable alteration in optic nystagmus after prefrontal lobectomy in monkeys, frontal and occipital lobectomies in dogs, occipital lobectomies in cats, and virtually complete decortication in rabbits.

Smith (367, 371) has reported that bilateral removal of the striate cortex spares optokinetic reactions in the cat, as does bilateral decortication of the guinea pig (368), for both real and stroboscopic movement of stripe stimuli.

Visser and Rademaker (410) found that decerebration in the pigeon failed to alter optically induced nystagmus.

Our interest here in the neural mechanisms underlying optokinetic reactions arises from the question as to what extent these same mechanisms are responsible for photic driving of the electroencephalogram and for critical-fusion frequency of intermittent light. The evidence which we have just reviewed points to subcortical mediation of optokinetic reactions. Our evidence from monkeys (cf. Monkey V), in which bilateral subtotal dorsal or ventral lesions of the striate cortex reduced photic driving of the electroencephalogram, and our evidence that c.f.f. is reduced in magnitude and variance by prefrontal-lobe lesions in man indicate cortical mediation of these processes. In line with this differential localization is an observation of Smith, Kappauf, and Bojar (371), who explored the optokinetic reactions of human subjects and of cats as a function of speed of movement of the stripe stimuli through the visual field. They concluded for their human subjects that optic nystagmus dropped out well below the critical-fusion frequency of the stripe pattern, which was illuminated with an average brightness of twenty millilamberts.

While the specific neurophysiology and the biochemistry of our P factor are as yet unknown, the various lines of evidence mentioned above strongly suggest its existence as a component of biological intelligence. The possible significance of the P factor to such fields as psychiatry, for example, is considered in chapter xi.

CHAPTER X

THE DIRECTIONAL FACTOR D

WE HAVE thus far considered evidence for the existence of three of the four primary factors specified by our factor pattern given in Table 4, page 41. We have interpreted the first of these, C, as a central integrative field factor; the second factor, A, as a basic ability for abstraction; and the third factor, P, as an undistorted power factor of the functioning brain. What is the significance of our fourth factor?

There remains an important parameter of biological intelligence which we have not previously considered. This has to do with the medium of exteriorization of intelligence, either from within or from without the individual.

At any given moment, intelligence is expressed or applied via a particular medium, be it writing or reading, listening or speaking, reasoning or thinking, music or painting. These are but examples from the gamut of human abilities which may be utilized at will. On the motor side each constitutes an organized motor outlet, a final common pathway. On the sensory side they comprise the mediums of experience and thus are basically perceptual. In either case it is possible to think of them as vectors of varying length, depending upon the magnitude or stage of organization of the associated special ability. Normally, all are present in a given individual but probably never to an equal extent. Some are more available than others for the exteriorization of intelligence. The basis for this variation is not yet clear. That genetic factors exert an influence, in some instances perhaps even a definitive influence, seems very probable. But it is also highly probable that the prepotent influence is usually contributed by learning. Genetic factors undoubtedly determine the general and special architecture of the brain, as in the case of other organs of the body. But learning determines the special organizations of its parts. The child must learn to walk upright, to speak, to write, etc. He must learn to read, although the neural structures for perceiving words have long since been present.

Our evidence for the existence of the directional or D factor in our factor pattern of Table 4, page 41, is not satisfactorily convincing. Only two of the thirteen indicators have loadings on this factor of .40 or higher. One

of these is indicator No. 4, the speed component of the writer's tactual-performance test, with a loading of .61. The other is indicator No. 19, the peripheral-vision component of the writer's dynamic visual field test, with a loading of .54. In one sense, the paucity of evidence for D represents a disadvantage, for most students of factor methods prefer that there be a minimum of three or four tests with substantial loadings on a factor before they will attempt interpretation. If we are correct in our interpretation of this factor as a modality factor, then the fact that eleven of our indicators out of thirteen have zero or vanishingly small loadings on D offers a distinct advantage. It would suggest, for example, that modality considerations played a secondary or negligible role in producing the total variance of our battery. Were this not the case, our image of the other three factors would have been reduced in power correspondingly, as their factor loadings were reduced, perhaps to a point at which our tentative interpretations would have been rendered impossible. The problem of measuring intelligence starts, methodologically at least, with the problem of testing beyond the modality influences. This is no less true where we wish to estimate the pathologies of intelligence, as in the brain-injured individual.

Analysis of the tasks presented in our two exceptions, indicators Nos. 4 and 19, reveals that in both instances there is present a factor of strangeness of modality. The tactual form board must be solved in three different trials while blindfolded. There is little doubt that deprivation of vision under these circumstances, forcing the subject to rely upon his tactual discriminations, results in considerable feelings of tension and awkwardness. These feelings are further accentuated by the mechanical necessity of holding one of the arms extended during the progress of the test. His first attempts at solution are but the merest gropings, made in an effort to gain a tentative comprehension of the realities with which he is faced—realities which he has never previously experienced through a visionless tactual medium.

A very similar situation prevails for the conditions of the dynamic visual field test. Here, too, the subject must direct attention to portions of the visual field which he normally ignores. He must do this at the same time that he is gaining information via his fovea which enables him to make microperceptual discriminations. Never before has he had to maintain a global orientation under test conditions. Even where the limits of his peripheral fields have been mapped clinically, i.e., on a tangent screen or on a perimeter, he had only to maintain a passive fixation of a foveal reference point and could direct his attention to the target as it was variously introduced into his peripheral visual field by the examiner. Un-

like the dynamic visual field situation, the target used in clinical mapping probably did not have a constant exposure time since the conventional techniques make no provision for this. This fact probably accounts for the relative insensitivity of clinical methods of mapping peripheral vision (206). It is of interest historically that the clinical discipline of ophthalmology continues to perpetuate this methodological error which first arose nearly one hundred and fifty years ago in the original efforts of Thomas Young to measure the limits of peripheral vision in man. The writer's d.v.f. test requires the detection of graduated peripheral targets exposed for only twenty milliseconds. The situation provides neither reward nor punishment as inducement to alter the level of performance.

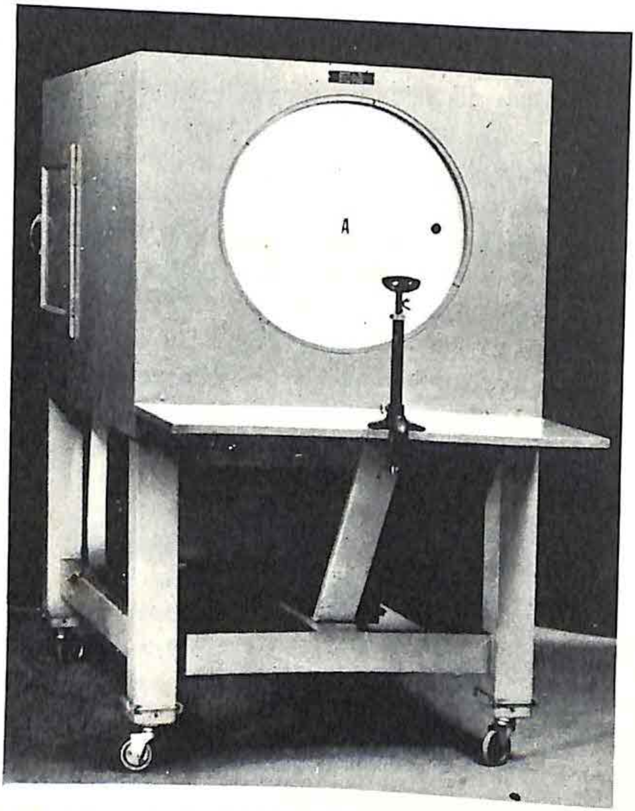


FIG. 10.—Halstead dynamic-visual field test (indicators Nos. 17, 18, and 19). Subject sits with chin in chinrest with eyes directed at center of screen. By pressing button switch, he exposes for 20 milliseconds a form and color target at center of screen and a graduated patch of light somewhere in the periphery of the screen. Subject records on prepared diagrams what was seen on each exposure. Machine adjusts automatically for each new exposure. The number of form, color, and peripheral-target discriminations correctly made is determined and scored separately as indicators Nos. 17, 18, and 19, respectively.

The subject remains ignorant of the objective accuracy of his performance on successive exposures. Thus, important conditions which are commonly regarded as essential for learning are lacking throughout. We have earlier interpreted this fact as being in line with the failure of this indicator to yield a substantial factor loading on our central integrative field factor C. Nevertheless, throughout three or four repetitions of this test on the same individual, the objective accuracy of performance may increase as if ordinary learning were occurring. This fact suggests that performance on the very first trial (the basis of the score for indicator No. 19) is influenced considerably by factors having to do with the modality or vector of experience, D. It is probable that the physiological threshold of peripheral vision is not reached during the first trial on the test. Only as the strange or initially awkward demands of the tasks are eliminated through repeated experience does the physiological threshold assume control of the variance.

The D factor and analysis of abilities.—Students of factor analysis have directed a great deal of attention in recent years to the analysis of special abilities. Is there a finite number of basic abilities from which all skills are somehow compounded? This is fundamentally the same question that was raised at the beginning of this chapter. The evidence from factor analysis studies seems to supply an affirmative answer, although the number of basic abilities involved is as yet by no means established. The available evidence has been collected for the most part from classroom populations or from other selected samples of the normal population. Validation of the abilities isolated rests at present upon statistical criteria. There have been no systematic attempts to relate such abilities to brain functions. Yet the field of human neuropathology offers great opportunities for such studies. Careful study of the pathologies of language functions produced by focal injury to the brain, for example, should provide a basis of validation in biological terms for abilities isolated by present-day statistical or other procedures.

Aphasia, agnosia, and apraxia.—Over a period of several years the writer has had an opportunity to study, with the aid of phonographically recorded examinations, several individuals with language disturbances produced by relatively focal brain injury. This material is properly the subject for a separate report and is referred to here briefly in the interest of directing attention to an important line of inquiry for the student of the biology of behavior.

Our observations suggest that aphasia is most commonly produced by injury to the left hemisphere in dominantly right-handed individuals and vice versa. It is probably fundamentally a disturbance in the form or pat-

tern of language and may be predominantly either receptive (impressive) or expressive (motor) in type or a combination of the two (215). When the area of brain damage is extensive, the patient may be practically mute in that he may use only one or two words to express himself. In one such patient, a high-school graduate, the total vocabulary employed during forty-eight hours following removal of a brain tumor was the word "ice-box." On the third postoperative day, a simple propositional sentence was uttered: "I want to go home." On the tenth postoperative day, a paragraph of sentences was expressed. From this point on, recovery of speech was rapid but was never complete. When examined four years later, the patient could carry on a simple conversation in a halting fashion but could not give the names of most commonplace objects, such as key, knife, fork, thread, and pencil, although she recognized each and could demonstrate its proper use. Our studies of this patient strongly suggest that the basic disturbance involved a partially reversible alteration of our central integrative field factor C.

The agnosias afford valuable evidence for the existence of the directional factor D; for in such individuals we find a loss of ability to recognize objects or symbols through a particular sensory avenue such as vision, hearing, or touch. The loss is here produced by focal brain damage which has not impaired memory functions, general intelligence, elementary sensations, or ability to execute simple to complex voluntary acts. The agnosias seem to be characterized by a disturbance in particular elements of language. In visual verbal agnosia, for example, the individual may be unable to read because he cannot recognize printed or written words, although he can recognize the individual letters of words. This condition differs from alexia, wherein the individual can recognize words but nevertheless has lost the ability to read. In either of these examples the individual may have no difficulty in recognizing and comprehending spoken words or sentences. He can use a telephone but cannot read his mail.

The apraxias likewise support our conception of the D factor as a component of biological intelligence as well as a conception of differentiated abilities. In purest form the apraxias represent loss of ability to execute particular simple or complex voluntary acts as a consequence of focal brain damage. The brain injury may have spared general intelligence and have produced no paralysis or inco-ordination of the related musculature. In agraphia, for example, the individual may have lost the ability to write because he can no longer execute the patterns of action necessary in forming individual letters of the written alphabet. He may be able to draw and to copy simple or even complex outline figures and to vocalize what he

would like to state in writing. He may have no difficulty in expressing language through any medium except writing. He may be an artist and yet be unable to sign his name to the picture which he has just completed.

A tentative conception of the organization of the language functions as inferred from our study of individuals with relatively pure forms of agnosia, sensory or impressive aphasia, motor or expressive aphasia, and apraxia is indicated schematically in Figure 11. As is indicated on the diagram, anomia, or loss of ability to recall words as names of familiar

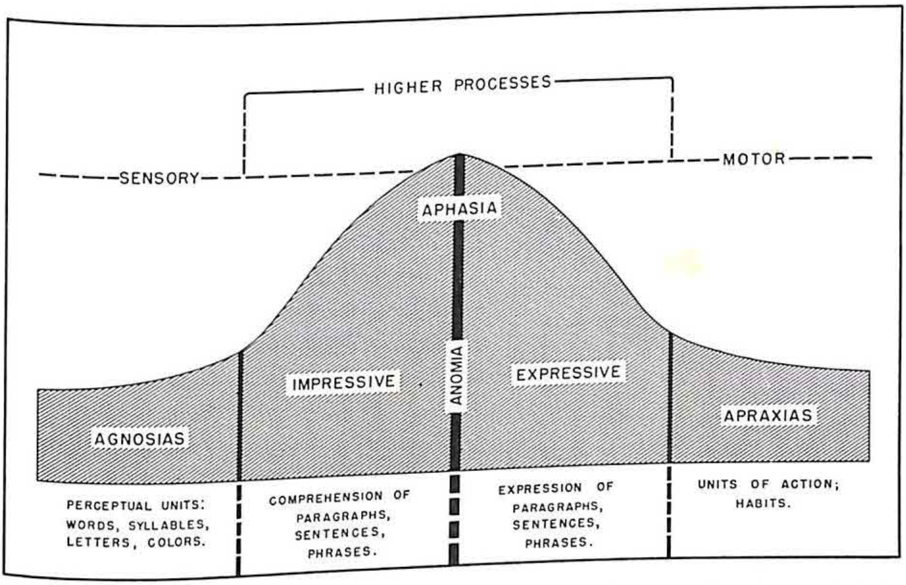


FIG. 11.—Schematic representation of relations of various language functions and their pathologies, as produced by focal brain injuries, to one another and to the higher mental processes. (See text for discussion of relationship of agnosias and apraxias to the modality factor D.)

objects or symbols, may be associated with aphasia that is predominantly either impressive or expressive in type. The solid black lines which cross the shaded portion of the figure are intended to represent transition points in the character of the behavioral disturbance encountered in patients studied by the writer.

It is to be hoped that some of the many cases of aphasia produced by the recent war are being brought under serious study in various countries; for, in view of the general advances in military neurosurgery, it is probable that many of these cases can yield evidence of far-reaching neurological, psychological, and psychiatric significance. One might go further and express a need commensurate with the general problems involved and sug-

gest that national registries of all brain-injured military and civilian cases willing and suitable for study should be prepared by appropriate personnel in the various countries and collected through a central agency such as U.N.E.S.C.O. Special research centers should be established in each country with adequate facilities for the application of diverse methodologies and techniques of investigation. And, finally, free exchange of scientific information and of investigative personnel from all relevant scientific disciplines should be encouraged by all appropriate means.

Intercorrelations of the basic factors.—Implicit in our conception of the D factor as a directional or modality factor is the notion that D would tend to be correlated to some extent with factors C, A, and P. If D represents

TABLE 10

INTERCORRELATIONS OF THE BASIC FACTORS

Factor	C	A	P	D
C	1.00	−.02	.24	.14
A	−.02	1.00	−.11	.27
P	.24	−.11	1.00	.28
D	.14	.27	.28	1.00

the medium of exteriorization of intelligence, then it will be operative whenever intelligence is operative. For the conventional mediums in the mature individual, we would expect D to control a negligible portion of the variance on a given measure of intelligence. Only where a new medium is employed for the first few times or where a conventional medium is employed under novel conditions would we expect D to assume significant proportions. Taking into account our method of scoring our tests as well as the nature of the tasks presented by each, we found indicators Nos. 4 and 19 (tactual performance speed and d.v.f. peripheral vision, respectively) to be the only tests in our battery which satisfy one or both of these conditions. In view of this fact we would expect our present battery to yield a small but positive correlation of D with the other factors. That such is the case is shown in Table 10, the values for which were supplied to me by Professor L. L. Thurstone. It may be noted that D is the only one of our factors which has a small and positive correlation with each of the other three factors.

CHAPTER XI

THE NUCLEAR STRUCTURE OF THE EGO

As we shall see in the following section, evidence from our studies of experimental anoxia in normal individuals and from our studies of localization of function in the brain supports our interpretation of the process factors C, P, and A and the directional factor D as the basic components of biological intelligence, which may undergo selective dissolution. In this sense it is believed that here for the first time the biological validity of basic factors in intelligence is demonstrated. There no longer would appear to be any necessity for psychology to go its own separate way, independent of anchorings in biological science (cf. 365). The dynamics of the process factors, as exteriorized through the D factors or specific abilities of the individual, seem to offer a workable key to that biological unity which is the healthy human organism. It is through these factors in the brain that the individual personality is modulated in its exteriorizations—to mediocrity, on the one hand, or to the highest reaches of human achievement, on the other.

We have arrived at a conception of the nuclear structure of the ego—a structure which is manifest in all cognitive processes. A structure which can be diagrammed in the same way that the chemist prepares a working diagram of a molecule. But before we turn to our diagram let us consider an aspect of behavior which has thus far resisted our attempts at measurement but which is an invariable concomitant of biological intelligence, namely, that of consciousness.

Heuristic value of consciousness as a variable factor.—The role of consciousness in animate behavior has acquired a long and occasionally even a distinguished history in scientific and in philosophical circles. Controversies arising from naturalistic approaches to the problem were especially marked during the latter half of the nineteenth century. The problem was brought to focus by the Pflüger-Lotze controversy over the question of whether or not the brain was the exclusive organ of consciousness or, more broadly, whether or not consciousness was coextensive with the nervous system. Lotze argued that the spontaneous movements of a spinal animal reflected the traces of learning, signs of an intelligence which is removed with the brain, existing "only in its after-effects." In addition to a great

stimulating effect upon experimental investigations, this controversy directed attention to the need for greater understanding of the nature of automatic actions.

Loeb (296), writing in 1900, recognized the importance of the problem of consciousness for physiology and psychology along with the need for improved instruments and methods of study:

> If brain-physiology gives up its fundamental problem, namely, the discovery of those elementary processes which make consciousness possible, it abandons its best possibilities. But to obtain results, the errors of the metaphysician must be avoided and explanations must rest upon facts, not words. The method should be the same for animal psychology that it is for brain-physiology. It should consist in the right understanding of the fundamental process which recurs in all psychic phenomena as the elemental component. *This process, according to my opinion, is the activity of the associative memory, or of association* [p. 12].

It is a matter of historical interest that, while Loeb saw clearly the need for understanding the biology of consciousness, he also prepared the stage for Pavlov's conditioned reflexology and Watson's behaviorism, wherein the problem and the phenomena of consciousness are conveniently omitted.

It is one thing to set forth the rubrics of a system as a system in however truncated a form. It is doubtful that chemistry, for example, could have long continued to obscure the phenomena of oxidation under the concept of phlogiston in the face of a rapidly developing science of physics. Conceptual errors in the physical sciences have been remarkably self-correcting, and herein lies a great strength. It is quite another matter for biology to develop a truncated conception of the human organism as the population unit of our complex social fabric. Unlike the physical sciences, biological and social sciences are in effect hierarchically organized in the modern world, and errors introduced below may ramify into social theory and applications. The biological view of the human organism must portray not only the present ecology of man but his potentialities for new ecologies, in short, his "becomingness." In civilized countries medicolegal and other institutions operate on the assumption of consciousness as an integral component of human behavior. Responsibility for criminal acts hinges even more upon "conscious intent" than upon knowledge of the law. It is possible, of course, that the principle of individual responsibility, a heritage from the ancient Greeks, is biologically unsound. The biologist has scarcely discharged his obligations when he merely ignores the problem. This view was clearly stated by C. J. Herrick (227), a neurologist, when he wrote in 1924: "The conclusion is that if consciousness, when present, is a real factor in the causative complex resulting in behavior, as I believe it

to be, obviously this factor cannot be ignored in the scientific analysis of the field of behavior as a whole" (p. 305). The argument, commonly encountered, that consciousness is a personal matter and, hence, is inaccessible to biological methodologies begs the question at issue. It is probably true that every consciousness is to some extent custom-built. This is true of my experience of the redness of a rose in comparison with the experience of any other individual. But such phenomenological facts have not stood in the way of considerable advances in our understanding of the chemistry and physics of color. Need it limit our approaches to the psychophysiology of color? This writer finds himself in agreement on this point with the view of Troland (407), who wrote in 1926:

> What is the exact chemical constitution and physical state of the substance in the brain which corresponds to "blue"? Or, conversely, what is the sentient quality which corresponds to the substance nitrobenzene, at ordinary temperatures? Such specific information is not at hand, and there seems to be no way in which we can arrive at it by logic or plausible guesswork. Nevertheless, it is entirely conceivable that progress in physiological psychology will yield this definite, detailed knowledge concerning the interrelations of consciousness and matter. There is no fundamental reason why we cannot trace the visual nerve impulses to their central destinations in the brain and discover exactly what happens there when blue is seen. This is a laboratory problem which cannot be solved in the author's arm-chair [p. 238].

Is consciousness an all-or-none affair?—Among biologists the view that consciousness is an all-or-none affair has found its principal support from investigations on animals of the effects of experimental concussion, electrical stimulation of the brain, experimentally induced syncope, and the specific action of certain drugs. But here the criteria for consciousness are necessarily gross, and it is extremely difficult, if not impossible, to scale degrees of consciousness.

Workers in the field of psychopathology, on the other hand, have long postulated for man a consciousness scalable into degrees or levels. In modern times this view has been set forth by Kraepelin (108), Freud (158), Jung (250), White (430), and others. Planes of anesthesia, planes of sleep, phenomena of hypnotism, somnambulism, hypnagogic states, deliria, dream states, disorientations of various types, and impairments of sensibility have been cited against the theory that consciousness is an all-or-none affair.

If we examine the psychological nature of the mental tasks which have been employed variously by psychologists in measuring "intelligence," great differences become apparent as to the demands in consciousness of the individual for successful performance. Between abstract thinking and simple addition in solving arithmetic problems or making simple pitch

discriminations there is a great step in consciousness. In the former the degrees of freedom of choice are almost without limit. As myriad patterns of content flash to the surface of "the stream of consciousness," some are "chosen," with the result that a *Principia* or a *Thermodynamics* which can include the principle of entropy is born. In the latter instances—addition and pitch discrimination—the degrees of freedom for choice are, in contrast, markedly restricted at the outset. The pitch of a tone is always (*a*) "higher than," (*b*) "the same as," or (*c*) "lower than" the comparison tone. Each step in the addition of 549 and 1,753, including the final sum, is either "correct" or "incorrect." Accordingly, the working level of consciousness is lowered to meet the demands of such tasks. This seems to be a fundamentally economizing process in mental performances. A corollary provides for the sparing of certain tasks in the presence of lowered levels of consciousness. One of the most striking illustrations of this process came to the attention of the writer in the course of some experiments on the behavioral effects of the anoxia associated with simulated high altitudes (195, 196) (see chap. xiii). A healthy, intelligent male subject was repeatedly exposed to a simulated altitude of eighteen thousand feet above sea-level in a decompression chamber. Among various behavioral measurements made on him was one involving the recognition or identification of airplane silhouettes serially exposed to his view for only twenty-thousandths of a second. As the chronic effects of repeated anoxia over a period of weeks became more pronounced, the subject complained that he could not "see" the figures as they were flashed on the screen. Yet, in spite of the most rigid experimental controls, he maintained a level of 100 per cent accuracy in his discriminations during this period. Under the stress of anoxia, the working level of consciousness was reduced almost to its lowest limit without sacrifice in test performance. At the same time, however, such abilities as abstract thinking, reasoning, social and moral judgment, were seriously impaired. The demands in consciousness of these tasks could not be satisfied under the experimental conditions.

The writer has examined several cases in which, following brain concussion, complex acts of behavior or even acts of high skill have been executed during an interval in which the individual did not "know" what he was doing. In one instance a nationally known professional baseball player played six innings of errorless ball after being "beaned" (struck on the head) by a pitched ball. He did not "come to" until nearly an hour after he had completed the game. In another instance a Catholic priest, with brain damage, played several holes of par golf with the writer during an interval of "total amnesia." A further illustration is provided from our

studies of anoxia. A well-known concert violinist[1] was studied for altitude tolerance in our decompression chamber prior to a tour by airplane of military camps in the Southwest Pacific zone. As one part of the examinations, phonograph recordings and movies were made as he repeatedly performed from memory a particularly difficult passage of a Bach sonata under experimental conditions which, unknown to him, variously simulated sea-level and the altitude of several thousand feet. Under conditions of acute anoxia, he became euphoric, mildly confused and disoriented, and presented other signs to attendant clinicians of impaired consciousness. Yet in the judgment of his professional accompanist and of the writer, based upon repeated examination of the phonograph recordings, his technical rendition of a most complex pattern of fingering on the violin was flawless under all the experimental conditions. The observation that certain qualitative aspects were lacking in some performances does not alter the fact that the formal aspects of a highly differentiated skill persisted without a continuously associated high working level of consciousness.

Another well-known violinist, Mr. Nathan Milstein, has described to the writer a concert, later repeated under more favorable circumstances, which, according to his accompanist, he gave without technical errors while virtually "asleep" from excessive fatigue. It is of interest that the creative artist never regards such automatic performances as works of art. One may speculate that the highly creative artist enjoys unusual freedom from his working level of consciousness—a freedom which is gained only when the technical demands of the medium have been mastered.

A highly skilled, professional juggler, examined by the writer, was found to have "embedded" an elaborate set of cues in his stage routine for "turning the audience on and off." "Turning the audience off" was for him putting the audience "out of mind" (consciousness), a condition necessary for his acts of highest skill. "Turning the audience on" was bringing the presence of the audience into his consciousness, a condition necessary for optimal stage rapport.

Relation of consciousness to the integrative field factor C.—We have found it convenient to limit our discussion of biological intelligence or ego in terms of our factor pattern presented in Table 4, page 41. It is extremely unlikely that our ultimate conception of the structure of biological intelligence will be satisfied with the four-factor space projected here. It seems highly probable to the writer, however, that the four factors which we

[1] The writer is indebted to Mr. Isaac Stern, to his accompanist, Mr. Alexander Zakin, and to Drs. Wright Adams and Henry T. Ricketts for their co-operation in this special study.

have isolated with the present battery of indicators are truly reflectors of brain functions. In this sense they are basic or planetary factors. But is it not reasonable to expect that, as we refine our indicators and develop additional ones, we will in time locate satellites and asteroids for each of the planetary factors? At such time our conception of ego structure will require *n*-factor space for its complete description. In the meantime we can only surmise that consciousness, for example, is an organized entity which bears a satellite relation to one or more of our four planetary factors. Of the four it is the writer's guess, set forth here only in the interest of urging basic neurophysiological and psychological research on the problem, that consciousness is a scalable entity which is closely related to our central integrative field factor C. It reflects the span of attention that is either operative or potentially operative in exploring the psychologically "new." It is probably influenced by the past experience of the individual as well as by the prevailing physiological state.

With the foregoing considerations in mind let us turn to our diagram of biological intelligence as presented in Figure 12. Our central integrative field factor C, which we have interpreted as representing the organized experience of the individual, is here diagrammed as the outer ring in the shape of a *C*. The open sector of this ring is narrow or broad, depending upon the demands in span of consciousness of the particular intellectual task. The working level of consciousness under ideal conditions is modulated directly by the "task." The situation is not unlike certain features of a modern radio receiver. Thus, C fluctuates much as the tuning eye of the receiver: When tuning is set to a broad band of frequencies for high-fidelity reception, the gap in the broken-circle pattern of the tuning eye is broad. When greater selectivity is desired and tuning is set to a narrow band of frequencies, the gap in the pattern is correspondingly narrow. While the fidelity is then said to be lower, discrimination of the receiver is higher and the "accuracy" of tuning is unchanged. Whatever the mental task, appropriate alteration in C is an economizing factor in performance. It helps to filter out interference or distraction and in this sense may be said to "protect" the task.[2]

Inside the broken ring are diagrammed the A-P-D factors. The A factor is a basic ability for abstraction or combining on the basis of a criterion. Biologically speaking, this ability or factor is an old one indeed. It is a vital property of the individual cell, perhaps nowhere more strikingly demonstrated than in the phenomena of mitosis. But, even before the cell,

[2] This neurophysiological variable is modulated by the task just as the pupillary and accommodative reflexes of the eye are controlled by the task.

it is reflected in the homogeneous principles of organization of certain crystal forms. Only the bases of pertinence are different in the grouping or combining of the mind. Temporally new properties of the inner and outer world become "equivalent" or "nonequivalent" conditions for the appearance of this or that form of behavior. On the side of sensory "equivalence"—where experimental analysis has thus far made most progress—it becomes increasingly apparent that these properties are phenomenal in character

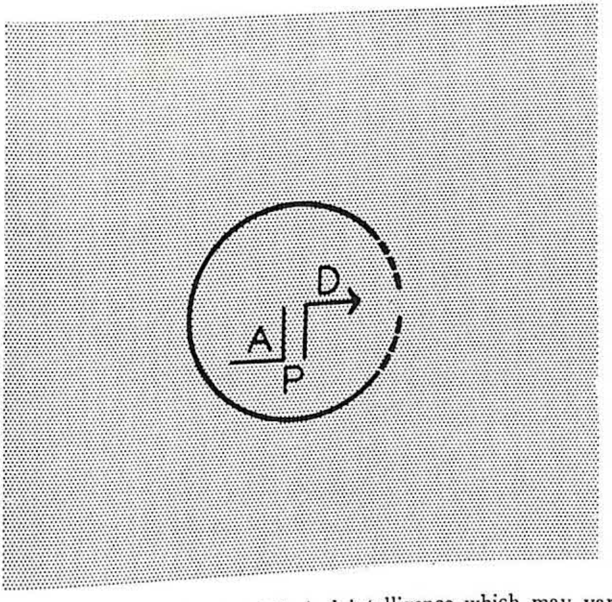


FIG. 12.—Conceptual model of unit biological intelligence which may variously be reduplicated throughout the cerebral cortex. Supporting evidence for the model from cytoarchitectural studies is thus far lacking. Outer ring in figure represents the central integrative field factor C. The A (abstraction), P (power), and D (directional or modality) factors are diagrammed as being interrelated and collectively related to C. (See text for description of these four basic factors.)

(162, 170, 171, 193, 270, 271, 273, 344). Klüver (271) has expressed the situation as follows:

> Whether or not behavior takes this or that direction is, generally speaking, dependent on whether or not this or that *phenomenal* property exists. The fact that something appears phenomenally as "red," "larger than," etc., cannot be deduced from a study of radiation or from the properties of the atom but only from studies of reacting organisms [p. 332].

Lashley (288) has stated a similar view:

> The adequate stimulus for an optic reflex can usually be described as a simple change in intensity of light, but the characteristic cerebral responses are based upon the distribution of intensities of light within the visual field. An analysis of the effective

> stimulus for such reactions regularly reveals a selective organization or abstraction of effective elements and a certain degree of generalization, even in the most primitive mammals. Thus, for the rat, as for man, visual impressions consist of organized objects, seen against a less coherent background. Discriminative reactions, when analyzed, are found to be based upon certain generalized features of the stimulus. Analysis of the properties of stimuli which determine reactions shows that in every case there is fundamental equivalence between a rather wide range of objects which have in common only certain general or relational characters, which cannot be reduced to terms of stimulation of identical nervous elements [pp. 301–2].

It seems probable, although the evidence available is by no means yet satisfactory, that neural integration involves progressive stages of abstraction, from the receptor through an intermediate center to the primary sensory field. But the phenomenal properties of the outer world conveyed to the various sensory fields of the cortex must in turn give rise to phenomenal neural events which likewise require abstraction for integration of the whole. It is this terminal stage in the neural train of events which corresponds with the A factor as isolated here.

The power or P factor is diagrammed as a connecting bond between the A and D factors in Figure 12. This is to indicate that at the level of intelligence these factors are indirectly energized by P. The P factor is a dynamic factor which, in terms of a single estimation, probably best reflects the over-all status of the brain. It is sensitive to the presence of relatively small lesions in the brain and to the effects of low-grade anoxia. There is reason to believe that it is also sensitive to certain concomitants of fatigue states (64, 364). Its specific physiology is unknown, but it would not be surprising should it be found to parallel those vital processes which sustain the brain and cortex at a high level of efficiency. The level of P probably fluctuates from hour to hour in many normal individuals and may manifest certain diurnal variations which parallel, if not produce, similar variations in psychomotor performance (cf. 364). In our quantitative results P appears to be disturbed by lesions occurring anywhere in the cortex (see Table 7, chap. ix), but especially by lesions of the prefrontal lobes. While it is not yet possible to make direct comparisons, it is of interest to recall that Lashley (286) found it necessary to postulate a general factor for t cortex of the rodent brain to account for the "equivalent" effects upon maze behavior of lesions placed in any part of the cortex. It is possible that progressive lowering of the P factor in the cortex was responsible for Lashley's findings for the rodent cortex and for Halstead's results for the avian cerebellum (188). In both instances, alteration of behavior was found to be proportionate to the size of the lesion regardless of location.

The D or directional factors are diagrammed in proximity with the P and A factors. Under limiting conditions, D represents a single ability

which is tapped by the particular task. The arrow is used to indicate D as the direction or outlet for the exteriorization of biological intelligence. Where it is desired to indicate the impressive or experiencing aspect of intelligence, D comes to stand for a "perceptual" factor, while the other factors are unchanged.

Limitation of diagram.—Most conceptual models have serious limitations, and that presented in Figure 12 is no exception. No means are provided, for example, for indicating the dynamic interrelations of the four factors. Yet in the exercise of intelligence these four factors are dynamically interrelated in the form of an organized unity or Gestalt. This Gestalt cannot be fractionated by introspective means. Only through the selective enhancement or depression of its component parts under special influences, such as brain injury or anoxia, may these components be detected with certainty. The diagram likewise does not indicate that each of the four factors may be scalable as to magnitude. Yet after appropriate studies it should be possible to assign weights to the various factors and thus to designate more accurately their relative strength in any given battery of psychological indicators. This might prove to be a useful procedure in the field of psychopathology, for example, where predictions concerning usable intelligence must be made. This possibility is discussed more fully in a following chapter.

Learning versus intelligence.—We have thus far considered our four factors from the standpoint of basic biological factors in intelligence. But it seems that they are also involved in the process commonly known as "learning." Lashley (286) found it necessary to emphasize certain similarities between learning and intelligence in order to rationalize a methodology for testing the rat. Here, faced with evidence drawn from study of the human organism, we find it necessary to emphasize certain dissimilarities which we must not permit our tentative interpretations of our C factor to obscure. In the first place, learning and intelligence appear to fall at polar extremes of the working level of consciousness. Whereas intelligence tends toward active extension of the field of consciousness, learning, as process, reduces the initial voluntary or conscious act through progressive stages to more or less automatic behavior. It is probable that this degradation process is accompanied by a lowering of requirements for amount of power (P) for effective exteriorization. It is thus that learning may function as a substitute for intelligence since the P requirements of the latter are always relatively high. In the new situation the generalizations established through prior learning make possible an immediate response whereas intelligence requires a conscious evaluation of the total field.

Second, intelligence and learning permit exteriorization of the personality withdifferent degrees of tension. Because it comes off immediately, the learned response is anxiety-free. Intelligence, on the other hand, by its very deliberateness, permits the mobilization of undischarged energy in the form of tension states or anxiety. Tension states have their neural concomitants which invade the field of consciousness. For intelligent behavior to come off, the P factor must be strong enough to hold these well in the background of consciousness. From the standpoint of predictions of human behavior, especially if we are to use practical measures of intelligence, it would seem to be desirable to measure two variables which are currently wholly neglected. First, there is needed a measure of tension mobilization or stress. Since the autonomic nervous system is directly involved in tension states, it is possible that the work of Darrow and his associates (100–103) may eventually reveal some component of the electroencephalogram which may serve as a practical index of tension. Second, the range of fluctuations in the P factor (and in C, A, and D) should be determined under basal and under stress conditions. A practical clinical test for P is already available in the form of the writer's test for critical-fusion frequency (indicators Nos. 3 and 3*a*, Appendix C).

A working model for psychopathology.—Science is an organized quest for constancies in nature. In so far as our model for nuclear structure of the ego reflects genuine constancies in intelligence, it provides a useful conceptual tool with which to order the unknowns of psychopathology. While the ego is always involved in psychopathology, are all components equally involved? What of the A factor in the schizophrenias, the P factor in the hypomanic and manic states or in the severe depressions, the C factor in the posttraumatic syndrome? These and many similar problems now become amenable to objective exploration with the results of such investigations specifiable in operational terms. Only the merest beginning—but, nevertheless, a beginning—has been made.

Let us turn now, in the following section, to the question of localization of the basic factors in the cerebral cortex of the brain; for if we should find evidence that these factors are differentially localized in the brain, then it may truly be said that we have isolated factors which are biologically or neuropsychologically significant.

PART II

LOCALIZATION OF FUNCTION IN THE BRAIN

CHAPTER XII

REPRESENTATION OF THE BASIC FACTORS IN THE BRAIN

IN THE Edwin Smith Surgical Papyrus (50, Case 9), translated by the late Professor Henry Breasted of the Oriental Institute of the University of Chicago and dated at approximately 1700 B.C., is recorded the earliest known case of injury to the frontal lobes (see Frontispiece). The patient evidently had suffered a "wound in the forehead producing a compound comminuted fracture of the skull." The case is of particular interest since the treatment employed may have reflected an association in the mind of the ancient doctor between the psychic disturbances of the patient and injury to his frontal brain, although in the opinion of Professor Breasted: "This case represents our surgeon's sole relapse into the superstition of his age, of which he has elsewhere so surprisingly divested himself. It was doubtless the resemblance of the frontal bone to a segment of the shell of an ostrich's egg, which beguiled some ancient physician into the belief that a paste made by triturating the shell of an ostrich's egg would be efficacious in healing a shattered frontal bone!" (50, p. 217).

Professor Breasted's translation of the section of papyrus reproduced in our frontispiece is as follows: "If thou examinest a man having a wound in his forehead, smashing the shell of his head, thou should prepare for him the egg of an ostrich, triturated with grease (and) placed in the mouth of his wound. Now afterward thou shouldst prepare for him the egg of an ostrich, triturated and made into poultices for drying up that wound. Thou shouldst apply to it for him a covering for physician's use; thou should uncover it the third day, (and) find it knitting together the shell, the color being like the egg of an ostrich" (pp. 219–20).

Over the wounded patient the following charm was to be said:

> Repelled is the enemy that is in the wound!
> Cast out is the evil that is in the blood,
> The adversary of Horus, on every side of the
> mouth of Isis.
>
> This temple does not fall down;
> There is no enemy of the vessel therein.
> I am under the protection of Isis;
> My rescue is the son of Osiris.

Of all the types of head-injury cases mentioned in the papyrus, this frontal-lobe case alone received recipe therapy; the others were given physical treatment, such as repair of depressed skull fractures. We may speculate, although the evidence is meager at best, that in this medical emergency of more than three thousand years ago a verbal form of psychotherapy was differentially applied by the physician to a patient with damage to his frontal lobes. We are denied knowledge of the outcome of the treatment in this interesting case.

The present century has brought forth a form of neuropsychiatric therapy no less interesting than that employed by the ancients. This therapy is known as "psychosurgery" or "lobotomy" and involves an operation upon the intact brain for the relief of psychopathy. The operation consists essentially of producing large subcortical lesions in both frontal lobes. Lobotomies, when studied before and after operation, and when compared with individuals in whom part of the cerebral cortex has been removed (lobectomies), make possible a re-evaluation of the problem of localization of function in the brain. With the evidence for the existence of basic biological factors in intelligence before us, we may turn once again to a consideration of the question with which our discussion began. To what extent is intelligence localized in particular parts of the brain?

In tracing the historical threads of the controversy surrounding this question in chapters ii, iii, and iv we found that there had been two almost completely independent lines of development. The first of these concerned the various attempts by psychologists to develop purely "psychological" theories of intelligence. Among the psychological theories of intelligence which have thus far been proposed are: (*a*) the unit-factor theory variously sponsored by Ebbinghaus (118), Binet (38), Terman (393), Stern (383), Franz and Gordon (151), Burt *et al.* (71), Stoddard (386) and Tredgold (406); (*b*) the two-factor theory proposed as early as 1904 by Spearman (378); (*c*) the three-factor theory urged by Holzinger (235); and (*d*) the multiple-factor theory, variously proposed by Thurstone (401), Thomson (395), and others. It was found that in the interest of clinical expediency such psychiatrists and psychoanalysts as Kraepelin (108), Bleuler (43), Freud (158), Janet (246), and Rorschach (349) have for the most part adopted some form of the unit-factor theory. It has not been possible to determine to what extent these theories are ultimately based upon genuinely different functions as opposed to mere verbal differences in exposition. None of them has ever been shown to have any foundation in neurology or physiology. Yet all of them have, to some extent, been useful or at least have been used as a sociological concept of

intelligence. Of especial interest is the fact that none of the foregoing theories makes specific provision for consciousness as a component of intelligence. In this respect they might just as readily apply to radar as to man. There can be little doubt that the ramifications of this omission in contemporary ethical and moral philosophy, for example, have been great indeed.

The second line of development concerned variously proposed neurological theories of intelligence. For the most part, these were found to be based upon impressionistic as opposed to objective methods for studying the behavioral effects of brain lesion in infrahuman animals. They included (*a*) the holistic theory, proposed by Flourens (143) in the first half of the nineteenth century and subsequently by Goltz (182), Ferrier (127), Loeb (296), Feuchtwanger (128), and Lashley (286); (*b*) the aggregation theory of Munk (328) and Von Monakow (318); and (*c*) the regional-localization theory, variously set forth by Broca (55), Hitzig (231), Flechsig (137), Franz (150), and Bianchi (36). It is not surprising that these theories have led to contradictions and controversies no less heated than those precipitated successively by the psychological theories mentioned; for, like the latter, they sprang from a priori assumptions as to the nature of human intelligence. Without a suitable methodology, the situations are few indeed in which the observation of a bit of behavior permits the conclusion that intelligence, as opposed, for example, to learning, has induced its appearance. This is no less true in investigations in which the possibility of "intelligent" behavior is nearly or completely eliminated, as in the case of the conditioned-reflex technique of Pavlov (333) and his followers.

A quantitative index of impairment.—From the standpoint of our general task, the results considered in the preceding chapters are heartening. Not only do the factor analyses confirm the existence of the basic factors isolated in our original investigations, they likewise support the notion that these factors are stable and universal attributes of human intelligence. But to what extent are these factors directly related to brain functions? Obviously, our answer to this question should include both the general and the specific relations of the various factors to the brain. Let us examine the general relations first. Our task would be simplified if factors could first be validated as a battery. By applying such a measuring device to individuals with brain injuries and to normal individuals, it would be possible to determine whether the factors are disturbed by brain injury. Should it turn out that they are in fact disturbed by brain injury, we

would have the direct biological validation which we seek. Such a measuring device has been employed in these studies.

Preliminary search for criterion scores.—For various reasons it was decided to use a relatively small group of neuropsychiatric patients in the initial attempt to establish criterion scores. Not the least important of these reasons was the consideration that there be available a complete medical history for each individual. It was also decided to use, as a reference group for brain-injury factor, patients convalescing from traumatic head injuries, since the clinical status of such patients could more readily be equated with that of the other patients in the group. Testing in each instance was done when the patient reached a stage in his convalescence at which he was ready for discharge from the hospital.

The group of twenty-three individuals finally selected as to age range and freedom from associated complicating pathology was known to be heterogeneous as to the nature of complaints or symptoms, history of a period of unconsciousness associated with a blow on the head, the period of time elapsing between such a blow and the beginning of testing, age and sex, the presence of verified brain damage, and the medical diagnosis. The group was known to include the following subgroups established by diagnosis:

I. A group with clear manifestations of psychoneurosis and without a definite history of head injury.

II. A group with a history of head injury and with evidence of psychoneurosis.

III. A group with a definite history of acute head injury with an interval of unconsciousness and with no clear evidence of psychoneurosis.

It was assumed that Subgroups I and III were relatively "pure" from the standpoint of diagnosis. It was also assumed that test criteria which selected the members of one of these two groups would ideally exclude all members from the other. Critical scores for each test of the total battery were then selected by inspection of the raw data. The effectiveness of each criterion in separating the "pure" groups was then evaluated by means of the chi square test (cf. Fisher [133]). The chi square test starts from the assumption that any two samples are random samples of the same material and then tests this assumption by showing whether they differ from each other more than may safely be attributed to random sampling or "chance." The various values of chi square were found to range from 0 to 4.05. This confirmed the impression gained from cluster analysis of the data that, while it was "highly probable" that some of the tests were not

differentiating between the "pure" groups, it was also "highly probable" that others were.

The tests yielding the ten highest values of chi square were then examined for differentiating power as a battery in three ways. First, the sum of the ten individual chi square values was determined. This sum was found to be 19.84. This corresponds to a value of P (ten degrees of freedom) of less than .04 (Fisher [133], Table III). Thus the odds against finding such differences yielded by the battery of ten tests, if they are due only to chance, are at least ninety-six to four, that is, twenty-four to one.

Second, the proportion of the battery of ten tests which satisfied the empirical criteria scores for the larger "pure" group was then determined for each individual. Expressed as percentages, these values were found to be 33, 11, 33, 22, 00, and 55 for Group I; and 88, 77, 100, 100, 100, 100, 50, 67, and 88 for Group III. (Deviation of these percentages from multiples of ten is due to incompleteness of data for some individuals.)

If a criterion of 67 per cent be taken for inclusion in Group III, only one patient out of fifteen is displaced with reference to the medical diagnosis in the "pure" groups. Since this patient, K. S., satisfied the criterion for Group I on the multiphasic personality inventory, it is possible that he is not a "pure" member of Group III and may actually belong in Group II.

Third, the multiphasic personality inventory revealed tendencies toward neurotic responses in four out of five individuals in Group I and in one out of six individuals thus examined in Group III. This would tend to support our initial assumption that these two groups are not in fact random samples of the same group. It would also tend to increase the empirical probability that the test results are reflecting a true difference.

The test results to this point may be summarized by stating that the odds are better than twenty-four to one that they are not due to chance alone and that further samples of patients with similar pathology would continue to be differentiated by the test criteria.

Reliability of criteria.—The next step involved a repetition of the foregoing procedures on another sample of patients corresponding to Groups I and III above. Fifty patients (twenty-five for each group) were selected from the neuropsychiatric service of an army general hospital. The clinical status of these patients differed from that of the previous parallel group in the sense that individual symptoms were more marked and were socially incapacitating for effective performance of army duties. The same indicators which were differentiating for the preceding group proved to be differentiating for this sample. In this instance, individual chi square

values were found to range from 0.2 to 19.1. For the ten most sensitive indicators the chi square values ranged from 4.7 to 19.1 and averaged 12.2. The differentiating power of this particular group of indicators was thus demonstrated at the 1 per cent level of confidence (where $P < .01$, the odds are less than one in one hundred that the result is due to chance [cf. Fisher (133)]). With these preliminary findings in the background, it was possible to proceed with confidence in the task of setting up an impairment index.

Theory of impairment index.—In addition to the presented statistical evidence that some of the indicators in the total battery were sensitive to brain-injury factor, the writer had had considerable opportunity to observe these indicators in patients with neurosurgical lesions. The statistical evidence was accordingly regarded as "in line with expectancy." But the question remained of how best to handle the measurements obtained from the standpoint of practical convenience. It was recognized that a single score or index which summarized performance on the selected battery of indicators would have great practical advantages. A well-known example of such an index is the I.Q., or intelligence quotient, which has come to stand as an index of performance over a range of diversified mental tests (393). The major objection to the use of such an index is that it averages out the peaks and troughs of ability and thus obscures these important details. An alternative procedure, long ago recommended and employed by Seashore (361), for example, in connection with his tests for musical talent, is to chart a profile for individual abilities. Visual inspection of such a profile reveals at once the relative strengths and weaknesses for a given subject. In many diagnostic and counseling situations the details reflected in a profile are of considerable use. There are other situations, however, in which an over-all score or index offers a real advantage for individual or group comparison. This is especially true where correlation techniques are employed. Furthermore, there is a strong tradition in medicine for the use of critical values or indices for diagnostic purposes. It may also be noted that a single over-all index represents the ideal of scientific parsimony since it is the simplest possible representation of multiple events or variance.

Performance on the remaining tests of the battery was in a sense important, for these tests are such that normal performance on them supplies control information concerning such variables as co-operation, attention, malingering, and level of effort. Furthermore, from the various personality tests in the battery certain "typological" information was obtained which seemed to be significant. In the interest of simplicity, however, it was

decided to construct an index to reflect brain-injury factor and for the present to regard the information yielded by the other indicators as supplementary.

The ten tests having the highest *t* value (i.e., differentiating power) for brain-injury factor were accordingly selected as the basis for an impairment index. In this arrangement an individual whose scores fall below the criterion scores on all ten of the key tests for brain-injury factor thus has an impairment index of 0.0; while, on a simple proportion basis, an individual who satisfies the criterion score on three of ten key tests has an impairment index of 0.3, or on all of the key tests, an impairment of 1.0. It is of interest to examine the results of the performance of the normal control subjects, from our larger control group, presented in Table 15, page 175. It will be observed that the impairment index is distributed in this group as follows: three subjects have an index of 0.0; seven have an index of 0.1; one has an index of 0.2; and three have an index of 0.3. No subject has an impairment index as high as 0.4. The average impairment index for the three control groups (see Appen. D) is 0.18. The average index for our neurosurgical cases as a group is 0.4, or more than twice as much. For those patients with a neurosurgical frontal-lobe lesion involving the cortex the impairment index averages 0.8, as shown in Table 16. By examining this table, we note that no frontal-lobectomy patient has an impairment index below 0.5, whereas the average index for nonfrontal cases is 0.26. The value 0.5 falls at the mid-point of the range of possible variance on the index scale. It is the point at which performance on 50 per cent of the key tests satisfies the brain-injury criterion. The fifty-fifty dichotomy is also the conventional standard for threshold values in a wide range of psychological functions, including sensory thresholds. In recent years this criterion has come to be employed as a basis for age placement of subtests in constructing tests of mental ability (185).

Distribution of impairment index.—The distribution of the impairment index in the heterogeneous subject population, two hundred and one cases used in the confirming factor analyses, is shown in the form of a histogram in Figure 13. It may be noted that ninety-seven cases (approximately 50 per cent) have an impairment index in the range from 0 to 0.4 while one hundred and four cases have an index in the range of 0.5 to 1.0. The envelope of this histogram is remarkably suggestive of a normal distribution curve.

An obvious objection to the foregoing interpretation of the impairment index is that the units on the scale are not equated in terms of their be-

havioral significance. That is, the ten key tests are not equally sensitive for brain-injury factor. There is also the consideration of differential localization in the brain of the functions reflected by the various tests. Furthermore, there is reason to believe that some of the tests are, to some extent, measuring the same aspect of the brain-injury factor. While these considerations argue against the concept of brain-injury factor as a threshold function, they do not contraindicate thinking of the scale as a reflector of probability that a brain-injury factor is present. In other words, the index, as now constructed, may be thought of as a statement of empirical

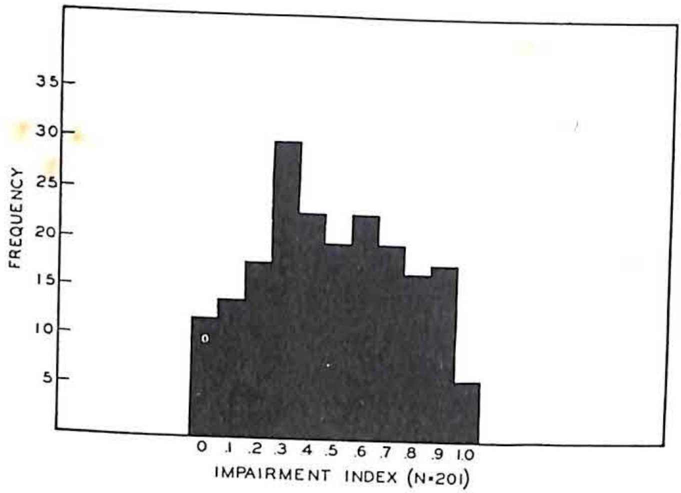


FIG. 13.—Distribution of Halstead impairment index in 201 individuals. Note suggested similarity to so-called "normal" or Gaussian distribution curve.

odds out of ten chances that a given individual has an impairment of cortical brain functions at the time of testing. This would appear to be a conservative view of the index, since, for a value of 0.5, the statement of odds would be that there are five chances in ten that brain injury is present while in actuality this value is sufficient to detect all our cases with verified brain damage to the frontal lobes. It is possible that odds of four chances in ten (index of 0.4) are clinically significant, especially if there is no evidence of a clinical depression or of schizophrenia in the individual and if performance on the control tests in the battery falls within normal limits. On the other hand, taken at its face value, it is doubtful that odds of three chances in ten (index of 0.3) can be thought of as clinically significant at this time for brain-injury factor, since three of our normal control subjects, Nos. 4, 12, and 13, equaled this value. In the case of

Subject 4, it is believed that the presence of a mild clinical depression (associated with the imminence of induction into the armed services) contributed directly to this relatively high index for a "normal" individual. Similarly, it is believed that the presence of acute "test" anxiety in Subjects 12 and 13 contributed directly to their relatively high indices. These are the youngest and the oldest, respectively, of our normal control group (ages fourteen and fifty).

CHAPTER XIII

THE BASIC FACTORS AND EXPERIMENTAL ANOXIA

THE practical problems arising out of the great expansion of aviation during the recent war have done much to stimulate research interest in the effects of anoxia upon the human organism. The many wartime research projects carried out both in this country and abroad have, in general, contributed detailed information concerning principles that had already been reasonably well established. These principles may be summarized for our purposes as follows:

1. Marked individual differences are to be expected in the reaction to the anoxia associated with high altitudes.

2. As far as is known, the physiological and psychological effects of acute anoxia, short of total collapse of the individual, are completely reversible.

3. Because of its high oxygen demands, the brain, and more specifically the cortex of the brain, is probably the organ in the body most sensitive to oxygen lack (7, 11, 82, 114, 135, 163, 194–97, 221, 302, 331).

Two experimental methods have been devised which are suitable for studying the behavioral and physiological effects of experimentally induced anoxia. One of these is known as the "re-breather" method (302) and involves the inhalation of dilute mixtures of oxygen and nitrogen. This method requires that the subject wear a facial mask or appliance and thus imposes serious limitations for certain types of behavioral investigations. The other method consists of placing the subject in a decompression chamber in which the atmospheric pressure (available oxygen) can be lowered at will by appropriate exhaust devices. Under favorable conditions, the chamber is constructed so that the subject may live as if in an ordinary room. This permits testing of his behavior over a wide range of functions.

Considerations 1, 2, and 3 are favorable for purposes of analyzing behavior. In effect, they make possible the study of selective loss and recovery of behavioral functions associated with experimentally induced reversible impairment of brain functions. By applying a battery of neuropsychological indicators to subjects under the well-controlled conditions of a decompression chamber, one should be able to ascertain which factors are the first to be disturbed by brain anoxia and which are the first to

recover when brain anoxia is relieved. Such studies would throw important light on results such as we have obtained from the examination of patients with neurosurgical (irreversible) lesions of the brain, for the latter always involve to some extent an abnormal individual whereas the method of experimental anoxia permits the study of reversible impairment of brain function in the normal individual. Any substantial agreement in the results of these two lines of inquiry would illuminate both. On the other hand, marked differences in the results could scarcely fail to be instructive. Fortunately, we are in a position to examine preliminary results, at least, of just such a parallel line of investigation.

As one part of a war-research contract between the Office of Scientific Research and Development and the University of Chicago (OEMcmr-113), initiated in 1942, on the acute and chronic effects of intermittent anoxia in man, the writer had the opportunity to study normal individuals before, during, and after exposure to experimental anoxia. Several indicators from the battery described in Appendix C were used for this purpose along with certain other indicators. The quantitative results were obtained in two stages. The first stage involved a general clinical and physiological survey of possible effects along with neuropsychological studies. These demanded special lines of investigation which were carried out on the same group of subjects by several different investigators. The second stage was designed to confirm a particular neuropsychological effect which emerged from the first investigation.

Initial investigation.—Several previous studies (7, 67, 70, 82, 114, 135, 163, 302, 362, 385) have shown that neuropsychological changes in the direction of decreased efficiency occur in the normal adult as the result of anoxia associated with altitudes over twelve thousand to fifteen thousand feet above sea-level. In general, however, such changes have not been found following brief or even prolonged exposure to altitudes in the neighborhood of ten thousand feet or lower. The absence of significant changes in test performance at these low altitudes has led to the rather generally established belief that for personnel of military age anoxia associated with altitudes up to ten thousand feet is innocuous. This conviction was reflected in former existing service regulations which required the use of O_2 equipment during daylight flights at altitudes above ten thousand feet.

Results.—Seven male subjects, ranging in age from seventeen to twenty-eight, were variously exposed in a low-pressure chamber to simulated altitudes ranging from ten thousand to eighteen thousand feet for four or six hours per day, six days per week. for a period of four to six weeks.

We shall limit our consideration of results here to those indicators which have been identified as measures of one or more of our basic factors. For a complete report on the work the interested reader is referred to the Office of Records of the Committee on Medical Research of the Office of Scientific Research and Development and to the writer's publications (194–97).

The central integrative field factor C.—It may be recalled that the indicator in our battery which had the highest loading on C (.58) was one of the Henmon-Nelson Tests of Mental Ability. This test was not employed in our studies of anoxia. It has been found to correlate in the neighborhood of .80 with the revised Stanford-Binet test for intelligence—and hence is probably reflecting much the same functions—which was used.

Alternate forms of the revised Stanford-Binet test were applied at the beginning and near the end of the period at altitude to the seven subjects. No reliable differences in test performance or in I.Q. were detected for any altitude, including that of eighteen thousand feet. If the Stanford-Binet test is a partial reflector of our central integrative field factor, which the writer suspects it to be, then that portion of C so reflected is not altered by chronic intermittent exposure to relatively high-grade anoxia in the healthy, adult male. In line with this interpretation is the fact that performance on our category test (indicator No. 2) was not disturbed at high altitudes, including eighteen thousand feet. This indicator has its major loading on the A factor, but it also has a substantial loading of .49 on the C factor.

However, certain observations were made during the course of the experiment which strongly suggest that the working level of consciousness was impaired at altitude although specific test performance may have been unaltered. A narrowing in the span of consciousness or attention, rather than a primary impairment of memory, may be the basis for the frequently reported lapses in memory for details of a particular task to be carried out under anoxia. Several competent observers have commented on their own experiences while under the influence of experimental anoxia. A feeling of drowsiness is one of the commonest early subjective signs of anoxia. The individual feels a need for sleep, although he may have had an unusually good sleep the night before. Combat pilots interviewed recently by the writer have informed him that this tendency toward drowsiness is not lacking within the zone of combat, where, in spite of regulations, much flying is done at fifteen thousand feet or higher without supplementary oxygen. One flight leader reported that on occasion over enemy territory he has fired tracer bullets toward planes in his own squadron in order to rouse their occupants to alertness.

In the altitude chamber this lowering of the level of consciousness may be reflected by neglect of an essential component in even a routine type of task. At ten thousand feet of simulated altitude, for example, the task of drawing samples of blood may seem to be extremely difficult for the investigator although he may have performed this operation hundreds of times before. He may prepare the tourniquet and neglect final adjustment of the syringe or prepare the syringe and neglect to release the tourniquet and so on. Many of the investigators associated with the present studies commented on this or similar difficulties. Early in the studies, while working at ten thousand feet, the writer nearly dismissed an assistant for insisting on turning a valve in the "wrong direction." The writer had previously operated this valve many times and knew that it had a left-hand thread. But he was certain that the assistant was wrong (actually, he was turning the valve in the proper direction). Only timely intervention from outside the chamber resolved the situation. Following this episode, the writer and his assistants used supplementary oxygen in all critical testing.

Flack (135) relates the experience of one observer in World War I who came back from a "highly successful" aerial scouting mission to discover that he had taken eighteen photographs of the same place.

The following personal communication is from a recent Swiss protocol of an experiment in which an accident occurred to the O_2 line of the observer, resulting in his collapse. Directly after the experiment the subject wrote the following account:

> At 3,500 m. [11,500 feet] I had mild sensations of a hang-over, remembered that we climbed further, and supposed that I fell asleep gradually. When I awoke I felt uneasy, wondered if all was going according to plan, and saw everything in twilight. All events were gradual, I did not see the observer. [The subject felt that this had lasted long enough. The observer evidently was doing nothing to end the experiment. All efforts and movements involved great difficulty.] I examined the dial in front of me [which regulated the controls], took off the mask, and pressed the "Descent" button when I slowly realized that the observer was prostrate—I looked around two-three times but everything was blurred—I pressed the button a couple of times and then said, "What's our altitude?" I thought the observer was delirious; his eyes were open, his expression blank, he was sprawled over two chairs, his face was mottled white and red and then he said, "Grab emergency mask." [The subject wrote further:] I had no sense of time, my field of vision was contracted, all my thoughts were concentrated on that button; I could not read the barometer. Tingling in my legs, headache in the front half of my head on descent.

Dunlap (114) as early as 1918 called attention to the fact that single tests of brief duration often fail to bring out any signs of alteration of performance under anoxia because "attention peaks" occur during which the individual is able to "pull himself together" and thus to simulate a good

performance. The writer has observed this phenomenon many times during chamber experiments. He has also observed that these "attention peaks" parallel an actual narrowing of the field of consciousness. The subject is not infrequently confused and disoriented at the same time that he is capable of fine sensory discriminations. A striking example of this was cited in an earlier chapter in the performance of Subject A. C. on the airplane-spotting test. Airplane silhouettes were presented tachistoscopically for only twenty-thousandths of a second in the form of a multiple-choice situation. At eighteen thousand feet Subject A. C. repeatedly denied ability to "see" the briefly exposed form, yet he made perfect discriminations throughout. Similar phenomena frequently have been observed by the writer while examining patients with traumatic or neurosurgical lesions of the brain.

In contrast to the alterations in level of consciousness under the influence of even low-grade anoxia, our quantitative results indicate that the A factor is relatively resistant to anoxia.

The A factor.—Our category test (No. 2) and our tactual-performance test (Nos. 5 and 6) were employed before, during, and after the period at simulated altitude in all seven subjects. If we refer to Table 4, chapter vi, it may be noted that each of these indicators has a substantial loading on the A factor. No significant alteration of the A factor by low-grade experimental anoxia could be detected by any of the three indicators. This observation is in line with our interpretation of the A factor as a biologically old factor. It is remarkably stable in character and, hence, is resistant to the early effects of anoxia. It is doubtful that this result can be accounted for in full by the "attention peaks" mentioned; for it is equally clear in the case of indicators Nos. 5 and 6, where incidental components of experience are tested for recall and accuracy of localization. No preliminary set or expectancy is established for these indicators, yet abstraction (A) is not impaired. However, the A factor has been observed to deteriorate at twenty thousand feet or above, and it is probable that it deteriorates at considerably lower altitudes in some individuals.

The P factor.—Indicators Nos. 3, 17, and 18 were used before, during, and after exposure in the chamber in some subjects. Again referring to Table 4, we may note that each of these has a substantial loading on the P factor. For technical reasons our data are incomplete for indicator No. 3. Evidence was obtained of a substantial lowering of the P factor in one individual, F. A., in many respects our most reliable subject. Indicator No. 3 is a measure of critical-fusion frequency (c.f.f.). During a four-week period at ten thousand feet, the c.f.f. of F.A. averaged from three

to four cycles per second lower than for the pre- and postexposure, ground-level controls. This difference is statistically reliable; his intratest variance never exceeded 0.9 cycles per second. This observation of lowering of c.f.f. under conditions of low-grade anoxia has been confirmed in a study carried out under United States Navy auspices but as yet unpublished.

Indicators Nos. 17 and 18, with loadings on the P factor of .64 and .61, respectively, are the central form and "color" components, respectively, of the writer's dynamic visual field test. They have loadings of .41 each on the C factor, but our other evidence indicates that this component is not primarily altered. The discrimination of these components is based upon exposure of the targets in the central visual field for only twenty milliseconds. Three out of seven subjects developed a marked and progressive impairment of ability to make these discriminations after about three weeks of intermittent exposure[1] at ten thousand feet. A lowering of the P factor is probably reflected here as a distortion or extinction effect, in the cortex, of concomitant brief stimulation in the periphery.

The D factor.—The most striking finding uncovered in the general investigation was revealed by indicator No. 19. This is the peripheral-vision component of the writer's dynamic visual field test. Referring to Table 4, chapter vi, it may be noted that this indicator has negligible loadings on the C, A, and P factors but a substantial loading of .54 on D. Beginning in the third or fourth weeks of exposure at ten thousand feet, three subjects developed a marked impairment of peripheral vision. This impairment progressed until the end of the exposure period and was not immediately reversible at the end of exposure. A similar effect appeared earlier and persisted longer in two subjects exposed at sixteen thousand and eighteen thousand feet, respectively. Because of the practical implications of these results, the effect for an altitude of ten thousand feet was explored in greater detail in the second investigation.

Peripheral vision, as measured in the d.v.f. test, appears to constitute an exception to the general stability of the D factor. Only further investigations will enable us to determine to what extent this component is primarily, or perhaps actually secondarily, impaired as a function of a general lowering of the power factor P.

Second investigation.—Seventeen additional subjects (eighteen experiments) were studied at ten thousand feet in a second investigation. Of

[1] Special studies with indicator No. 23, an anomaloscope, have revealed that color vision per se is not impaired in individuals who develop an impairment of the dynamic visual field under low-grade anoxia. Likewise, alteration in visual acuity per se is probably not responsible for the deterioration in form discrimination, since performance of these same subjects on Hecht's Contrast-Discrimination Test (221) was not impaired.

these, ten developed a marked impairment of the peripheral component of the d.v.f. during the period at altitude, while seven did not. One subject, J. M., who had shown a marked impairment of the d.v.f., was re-exposed eight months later. During the second exposure he was given 100 per cent O_2 for one hour at the mid-point of each daily five-hour period at altitude. Under these conditions, no impairment of the d.v.f. developed. Including our earlier results, thirteen (65 per cent) of twenty different subjects developed the visual impairment at ten thousand feet. With one exception (at eleven thousand and five hundred feet), all our subjects studied above ten thousand feet developed the impairment. Representative curves for indicator No. 19 are shown in Figure 14.

Relationship to alveolar gas tensions.—Serial determinations by Dr. Wright Adams of alveolar gas tensions, by the method of Haldane and Priestley, were made on sixteen of the twenty subjects exposed at ten thousand feet for each visual test day. No relationship was found to exist between impairment of the dynamic visual field and O_2 or CO_2 tensions under conditions of low-grade anoxia. This is in contrast to the gross relationship suggested by the findings above ten thousand feet, where the visual impairment was more marked, appeared earlier, and was reversed more slowly, as the degree of anoxia was increased.

Relationship to body chemistry.—The four subjects exposed at altitudes above ten thousand feet were the same subjects used by Bryan and Ricketts (67) in their search for possible effects upon the adrenal cortex of chronic intermittent anoxia. These subjects were maintained on constant, weighed diets; and careful metabolic technique was followed throughout. Studies were made of glucose tolerance and of urinary excretion of sodium, potassium, chloride, phosphorus, nitrogen, and the 17-ketosteroids. A marked impairment of the dynamic visual field was found in all these subjects during their period at altitudes ranging from eleven thousand and five hundred to eighteen thousand feet. However, with the possible exceptions of a slight increase in potassium excretion in two subjects, a transient rise in 17-ketosteroid excretion in one subject, and a moderate decrease in tolerance for glucose in two, the findings of Bryan and Ricketts were negative.

Relationship to general behavior.—It is difficult to estimate the response of individual personalities to the tedium of confinement in chronic experiments of this type. By good fortune the general health factor was excellent throughout the experiments. The subjects were paid at an hourly rate during the total period of participation. With minor exceptions, their cooperation was excellent. Some who developed marked impairment of the dynamic visual field showed actual improvement on certain other tests of

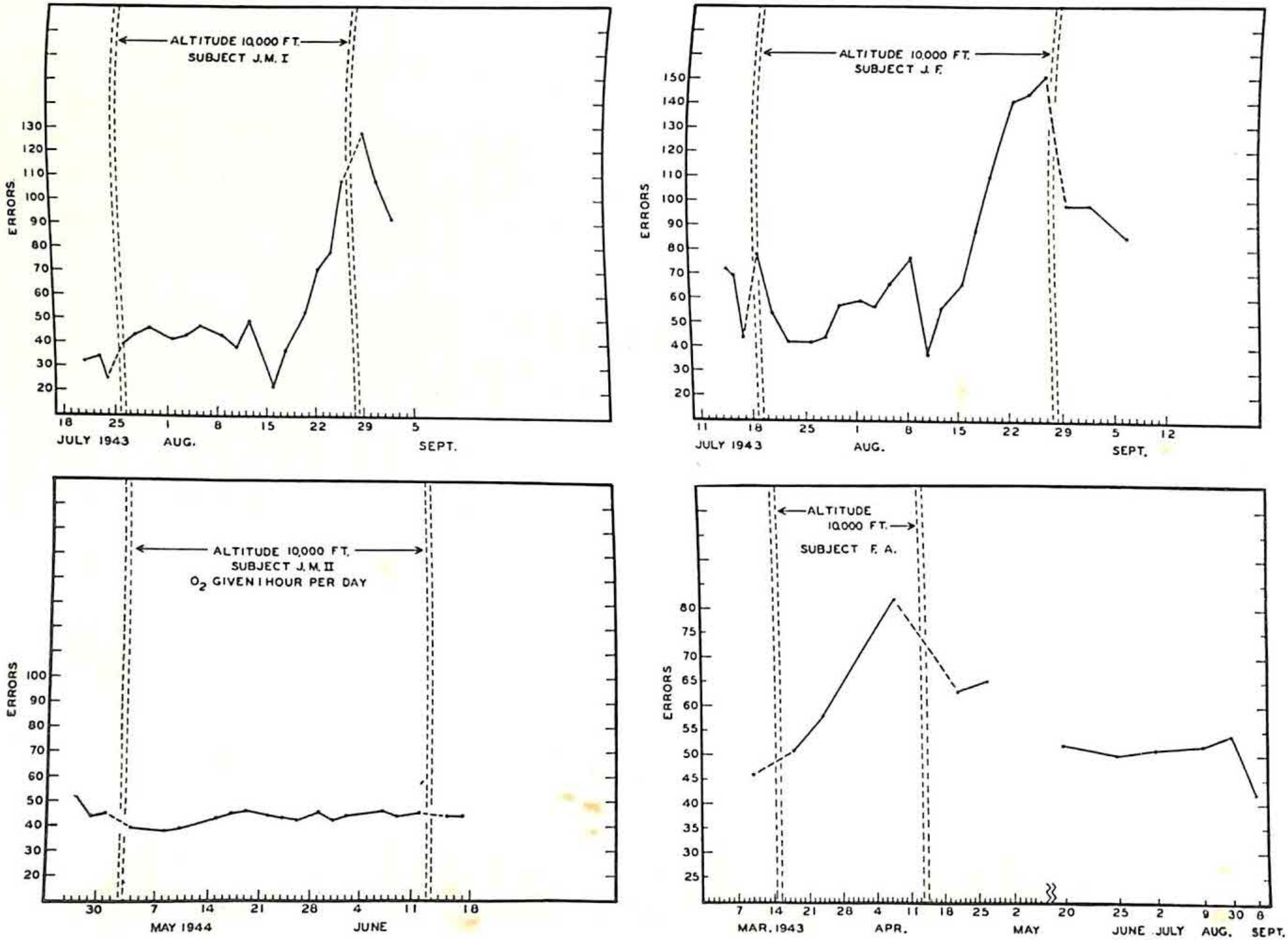


FIG. 14.—Impairment of peripheral vision (indicator No. 19) following intermittent exposure to anoxia in a decompression chamber over a period of weeks in healthy male subjects. Note that impairment did not develop when subject (J. M. II) was permitted to breathe 100 per cent oxygen during the middle hour of the daily five-hour exposure period.

the paper and pencil variety (cf. Fig. 15). All of them developed minor complaints during the period at altitude, however, or manifested minor clinical signs of altered behavior. These ranged from "mild headaches," "mild blurring of vision," "drowsiness," "prickling sensations on the skin," and "slight general motor weakness," to periods of mild euphoria or depression, restlessness or lassitude. It is of interest in this connection that the various investigators concerned with the total project independently

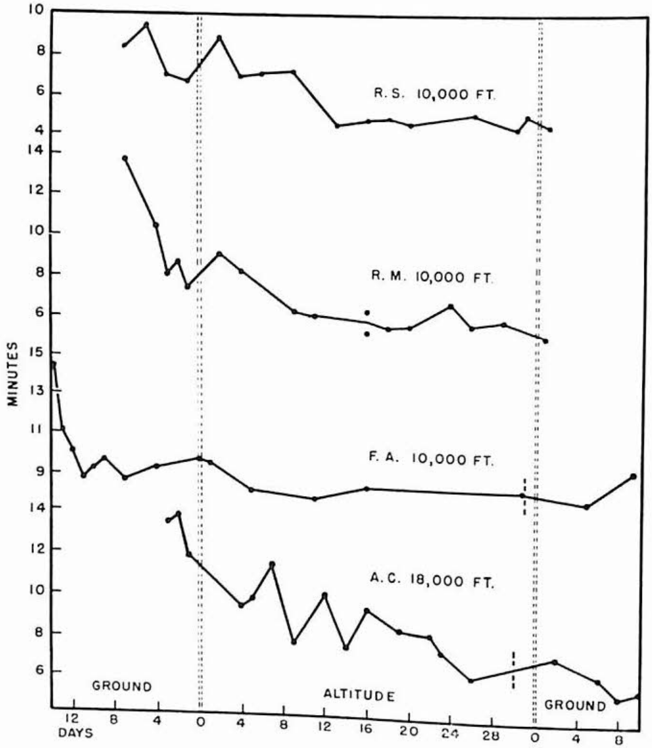


FIG. 15.—Speed of performance in finding correct solutions of navigation problems for healthy male subjects variously exposed to the anoxia of simulated altitudes. Note little change in performance at altitude in subject F. A. and improvement in performance of other subjects.

reported subjective symptoms of an "effort syndrome" which tended to become more marked with the repetition of the task of measuring or obtaining samples at ten thousand feet. It is significant that peripheral vision was altered in some subjects without a concomitant alteration of central vision for the low-contrast forms in the test exposed for only twenty milliseconds. Discrimination of these forms at all under the conditions imposed by the test in effect guarantees good co-operation on the part of the subject.

As in the case of alveolar gas tensions, no clear relationship could be

established between the visual effect and renal vascular changes (Dr. Alf S. Alving), cardiovascular changes (Dr. Emmet B. Bay), blood chemistry (Dr. Guzmán E. S. Barrón), psychiatric changes (Dr. Hugh T. Carmichael), electroencephalograms (Dr. Theodore Case), peripheral blood flow (Dr. Milton Landowne), or the metabolic effects (Drs. Henry T. Ricketts and A. Hughes Bryan).

Significance of the dynamic visual field test results.—The d.v.f. test is a contrast-discrimination test involving both the central and the peripheral portions of the visual field. In this investigation we have been primarily interested in the responsiveness of the peripheral field to anoxia, and it is this component which is analyzed graphically in Figure 14. Errors represented in the graphs indicate that targets that were presented in some part of the peripheral field were not seen by the subject. They thus reflect an alteration in sensitivity or a constriction of the d.v.f. The impairment revealed by this test is effectively a form of blindness in no way revealed by conventional eye examinations. It is of particular importance that this form of blindness develops insidiously while the subject is exposed intermittently to altitudes as low as ten thousand feet. Our subjects were completely unaware of their altered performances.

The mechanism underlying this visual impairment is by no means clearly understood, nor can it be stated with certainty at what level in the visual system the impairment arises.[2] The stability of the performances on the test from day to day in such subjects as J. F., J. M. I, and F. A., who developed impairment (Fig. 14), and in subject J. M. II, who did not (Fig. 14), indicates clearly that such factors as "fatigue," "lack of co-operation," or "inadequate motivation" are not directly responsible for the impairment. On the other hand, the absence of direct relationship between the impairment and alveolar gas tensions suggests that a secondary process is set in motion as a response to anoxia in some individuals. It is this secondary process which perhaps in turn gives rise to the delayed but progressive impairment and delayed recovery which we found to be reflected as an impairment of the dynamic visual field. Whatever the mechanisms underlying the impairment may be, it seems probable that their elucidation will eventually clarify not only the general and specific aspects of acclimatization to anoxia but the neurophysiological nature of the power or P factor and possibly other factors which we find to be basic in biological intelligence.

[2] The impairment might be rising in the retina, the geniculostriate system, the primary optic cortex, or a secondary cortical projective system en route to consciousness. Mechanical obstruction by pupillary constriction is eliminated from consideration since the impairment is not found by conventional methods of perimetry at a time when it can be detected by the d.v.f. test.

CHAPTER XIV

IMPAIRMENT INDEX AND THE FRONTAL LOBES

We have considered evidence of two kinds that our basic factors in biological intelligence are represented in the brain. First, it was shown that an impairment index, comprising the best available measures of the basic factors, may differentiate between verified brain-injured neurosurgical patients and nonneurosurgical patients (controls). Second, evidence was presented to the effect that experimental anoxia in the normal, healthy adult male produces a selective disturbance (and recovery) of the basic factors. In physiological terms, experimental anoxia produces a reversible impairment of brain functions. Thus, both lines of evidence point to a representation of the basic factors in the brain. Let us now see to what extent they are represented in particular parts of the brain. We may phrase our problem somewhat more specifically in the form of a question:

To what extent does our impairment index, which is composed of the C, A, P, and D factors, reflect functions which are localized in the brain? The data from our neurosurgical cases provide a clear answer.

In Table 11 are shown the impairment-index values obtained for forty-five cerebral lobectomies, two cases of subdural hematoma, marked with daggers, and eight frontal lobotomies. The index value of 0.9 for Case 50, a bilateral frontal lobectomy, is entered in both columns for left- and right-sided lesions to avoid undue weighting. The index values obtained for the fourteen normal individuals from our larger control group have been included for comparison.

Statistical test for differences between groups.—We may again apply a conventional statistical criterion in appraising the significance of the differences in the averages shown in Table 11. Where probability (P) is less than .05, a significant difference in two means is indicated. In Table 17, Appendix D, are presented the values of P for our impairment index and for each subtest on which it is based for our various groups of subjects. With the exception of indicator No. 16, which yields a value of $P < .016$, all the subtests in our impairment index yield values of $P < .001$ between our control group and our group of frontal lobectomies. Likewise, the impairment index reflects a true difference between these two groups;

$P < .001$. Between the frontal lobectomies and the nonfrontal lobectomies, once again $P < .001$; while between the control group and the nonfrontal lobectomies, $P < .002$.

Inspection of Table 11 reveals several facts of interest. In the first place, no frontal lobectomy, either left- or right-sided, has an impairment index below 0.5. The average index for left-sided frontal lobectomies is 0.86, while for right-sided ones it is 0.78. This difference is not significant.

TABLE 11*

IMPAIRMENT-INDEX VALUES OBTAINED FOR NEUROSURGICAL PATIENTS ACCORDING TO LOCATION OF BRAIN LESION

Frontal Lobe				Temporal Lobe		Parietal Lobe		Occipital Lobe		Cerebellum	Normal Controls
L.F.	R.F.	Lobotomy Pre.	Lobotomy Post.	L.T.	R.T.	L.P.	R.P.	L.O.	R.O.		
1.0	.9	.2	.1	.2	.2	.2	.4	.2	.2	0.0	.0
1.0	.6	.3	.4	.3	.1	.1	.2	.0			.0
.8	.6	.3	.4	.2	.0	.6		.1			.1
.7	.9	.4	.7	.2		.7†					.3
1.0	.5	.4	.4	.9							.1
1.0	.6	.9	.8	.8							.2
.9	.5	.7	.7								.1
.7	1.0	.8	.8								.1
.7	.8										.0
1.0	.8										.1
.9†	1.0										.1
.8	.8										.3
.7	1.0										.3
.9‡	.9‡										.1
Av.... .86	.78	.50	.55	.43	.10	.40	.30	.10	.20	......	.13

* Original data are given in Appen. C.

† Extra-dural hematoma (index of .2 obtained following surgical intervention).

‡ Bilateral prefrontal lobectomy.

In striking contrast, the average index for nineteen nonfrontal cerebral cases is 0.26, and for fourteen normal control subjects it is 0.13. Expressed in another way, the mean impairment index for our frontal lobectomies is about six times that for our normal control subjects and about three times that for our nonfrontal lobectomies. Thus, there can be little doubt that our impairment index is reflecting brain functions and, more specifically, functions which have their maximum representation in the frontal lobes of the brain.

Subcortical lesions (lobotomies).—Prefrontal lobotomy, or focal brain surgery, for relief of some forms of psychopathy has passed its first

decade. On November 12, 1935, Egas Moniz, a Portuguese neurologist, and Almeida Lima, a surgeon working in co-operation with Sobral Cid, a psychiatrist, undertook the treatment of psychotic patients by surgical interruption of the frontal association pathways in the brain. The favorable result, with confirmation, was reported immediately in several brief communications (319–21). A monograph by Moniz, describing the results of this operation in twenty cases, appeared in June, 1936 (320). This monograph attracted the attention of Freeman and Watts (152), who performed the first prefrontal lobotomy in this country about three months later on September 14, 1936. Following their favorable report, the method was soon taken up by others. It is probable that nearly two thousand such operations, with variations, have been performed in this country during the past decade. More than one hundred references to leucotomy or lobotomy, as the operation is now known, are to be found in the literature (see Bibliography in 202).

General results reported for prefrontal lobotomy.—In a monograph on the subject published in 1942, Freeman and Watts (154) summarized their general results for a group of eighty cases as follows: "In our opinion, 63% of the cases have resulted satisfactorily, while in only 14% of the survivors can the results be considered bad, either from the standpoint of a return or persistence of symptoms or from the standpoint of antisocial behavior that makes the individual a difficult problem in his environment" (p. 287).

Ziegler (439), in 1943, surveyed the results to date for six hundred and eighteen lobotomies from one Canadian and seventeen American centers. The results of this survey, without reference to the preoperative psychopathy, are shown in Table 12.

Examination of these reported results, essentially as classified by Ziegler, reveals an outlook no less optimistic than that of Freeman and Watts. Thus, 66.2 per cent of the cases are reported as showing marked improvement or social recovery; 83.8 per cent, slight or better improvement; 10 per cent, no change; 6.1 per cent, still less favorable results. Unfortunately, the validity of these findings is impossible to assess, except perhaps as vital statistics. At no point have other than superficial attempts been made to standardize the criteria for the preoperative and postoperative clinical status of the patients. Not a single patient has been adequately studied. For the social responsibility to do this there has been substituted a phenomenal array of case statistics. The pyramiding of unknowns, however, is scarcely a pathway to knowledge.

This is no less true in those few instances in which clinical opinion has

been supplemented by psychometric devices (cf. 154, 238, 239, 264, 345, 346, 391). In no instance has the psychological test or battery of tests employed ever been shown to be sensitive for frontal-lobe functions. In several instances just the opposite has been true. We may use a particular test as an illustration. Many other tests employed are either standardized in terms of it or highly correlated.

For the Stanford-Binet test, widely used as a measure of psychometric intelligence, the range of postoperative I.Q.'s for unilateral and bilateral frontal lobectomies reported in the literature is from 54 to 152, with a mean value of 108. For cases examined preoperatively as well, an average drop of

TABLE 12*

GENERAL RESULTS OF PREFRONTAL LOBOTOMY IN 618 CASES

	Cases	Percentage
Clinical status:		
Recovery	215	34.8
Markedly improved	194	31.4
Slightly improved	109	17.6
Unchanged	62	10.0
Worse	8	1.3
Death (operative)	12	1.9
Death (subsequent to operation, including two by suicide)	18	2.9
Occupational status:		
Working part or full time	251	42.7
Discharged but unable to work	60	10.2
Hospitalized	277	47.1
Unknown	30	....

* Data taken from Ziegler (439).

one point in I.Q. has been found postoperatively, the range being from a loss of fourteen points to a gain of eleven points. Since bilateral (and possibly even unilateral) frontal lobectomy represents more extensive ablation of brain tissue than lobotomy, it would appear unlikely that a test known to be insensitive to the former would prove to be sensitive for the latter. It might be commented in this connection that a physicist who purported to measure microvolts with a voltmeter would scarcely be taken seriously.

Lobotomy, it seems, involves damage primarily to subcortical structures and leaves the cytoarchitectural areas of the cortex relatively undisturbed. This is the opinion expressed by Freeman and Watts (154) following their examination of autopsy specimens:

> In conclusion it may be said that prefrontal lobotomy cuts across the axones of cells situated in the nucleus medialis dorsalis of the thalamus and causes atrophy of the

> cells without in any appreciable way affecting the anatomic organization of the cerebral cortex. It may be further concluded that the symptoms from such a lesion are traceable to the thalamic or emotional component rather than to the disturbance in cortical or intellectual function. Finally, since the frontal cortex is intact, we conclude that the cells are maintained in biologic activity by the persistence of collateral and association fibers that are not injured by the operative procedure [p. 191].

They have not studied the results of decorticating the frontal lobes, the effects of removing both the cortex and the subcortical structures, as in frontal lobectomies, or comparable lesions outside the frontal lobes. Hence, their speculations about the functions of the frontal lobes can be accepted only as such.

Through the courtesy of the Illinois Neuropsychiatric Institute,[1] it has been possible for the writer to examine several patients before and after prefrontal lobotomy (202). The quantitative findings on these cases are of especial interest here since they help to answer the question as to what extent our basic factors, C, P, A, and D, are disturbed by subcortical lesions. In some instances, the clinical status and the attitude of co-operativeness toward the tests were such that excellent preoperative determinations could be made before the intact brain had been surgically damaged.

Impairment index and sub cortical lesions.—The impairment indices obtained preoperatively (gray crosshatching) and postoperatively (solid black) in these cases are shown as a histogram in Figure 16.

It may be noted that there is no evidence of a reliable or consistent shift in the impairment index which may be attributed to lobotomy. In only one instance (M. C.) did a shift of more than one point on the impairment-index scale occur following the operation. The preoperative index for M. R. is unknown. Each patient was examined not less than forty-two or more than ninety days after the operation. In some instances, the case was followed by serial examinations over a period of about three years. Little change in the impairment index was found. Thus, in contrast to the findings for frontal lobectomies, biological intelligence, as reflected by an impairment index, does not appear to be altered significantly by prefrontal lobotomy. A possible reason for this result lies in the nature of the impairment index. Each point on the scale stands for performance on an indicator which has been found to reflect brain damage in neurosurgical patients. The scale thus reflects probabilities in chances out of ten (points) that the individual subject has performed as do individuals with known brain damage. No frontal lobectomy has been found to score below 0.5 on

[1] The writer is indebted to Drs. Francis J. Gerty, Hugh T. Carmichael, Percival Bailey, and Paul C. Bucy for their co-operation.

this scale. Examination of the preoperative index values suggests that biological intelligence was impaired prior to operation in some cases and that lobotomy neither relieved this impairment nor consistently increased it.

These findings bear significantly upon our other evidence concerning localization of function, especially in view of the fact that prefrontal lobotomy is primarily a subcortical operation and, apparently, leaves the cytoarchitectural areas of the cortex essentially undisturbed (154). In

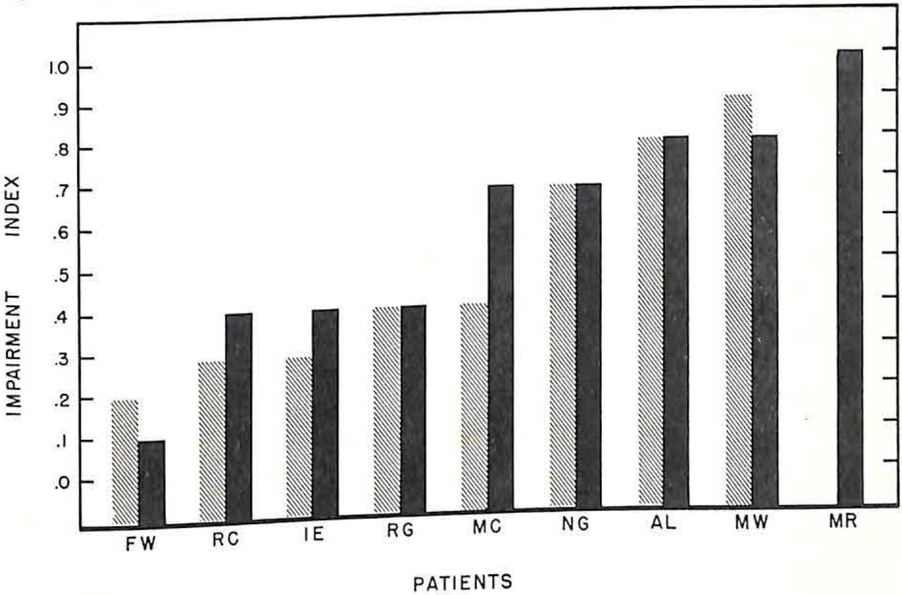


FIG. 16.—Preoperative (*gray*) and postoperative (*black*) values of Halstead impairment index in frontal lobotomies. Note that a substantial degree of impairment was present in some patients prior to, as well as after, operation and that F. W., for example, does not manifest the degree of impairment found for every case of frontal lobectomy.

one of our cases (F. W.), and in three others to a lesser extent, the pre- and postoperative index values are significantly less than those yielded by frontal lobectomy wherein the cortex is removed along with the subcortical white matter. This indicates that the functions reflected in the impairment index are maximally represented or localized in the frontal lobes and, more specifically, in the cortex of the frontal lobes.

The relation between the impairment index and the extent of injury.—The range in the surface extent of the lesions in our neurosurgical cases is from 0.67 to 32.5 per cent of the total surface area of the cerebrum, with an average of 8.3 per cent. In determining these values, use was made of the writer's brain-lesion charts, a schematic brain model, a lateral x-ray plate

for each patient, and actual measurements of the lesion during the terminal stages of the operations. The details of the technique for charting the lesions are given in an earlier publication (201). The charts are similar to those shown in Appendix B of this volume. Each chart consists of four carefully prepared[2] diagrammatic views of the brain reduced to one-third of life-size (a superior, a lateral, a ventral, and a median-sagittal). Measurements of the lesion made at the time of operation are translated to scale on these four standard views. A percentage (brain size) correction factor is calculated from constants taken from the patient's lateral x-ray plate and the similar view on the schematic brain model. The area of the surface lesion in each view of the chart is then determined directly by means of a planimeter.

In Table 13 are shown the correlations obtained between the percentage of surface lesion and the impairment index. For all neurosurgical cases r is $-.091$; for frontal cases r is $.090$; for nonfrontal cases r is $.162$. None of these values is reliably different from zero.

Our material is not suitable for examination of the degree of impairment and extent of destruction within different cytoarchitectural fields. The technique of charting the lesions does enable us to raise this question for the gross surfaces of the brain. For the relation between extent of lesion on the superior surface of the brain and impairment r is $.096$; for the lateral surface r is $-.291$; for the ventral surface r is $-.156$; and for the medial surface r is $-.116$. Again, none of these values is reliably different from zero.

Possible sources of spuriously low correlations.—An obvious source of possibly spuriously low correlations lies in the measurements of the lesions. The technique attempts to project onto plane surfaces lesions distributed on curvilinear surfaces. Errors of relatively unknown magnitude are thus introduced. By use of a standard technique of charting the lesions, a substantial portion of the error of measurement is undoubtedly reduced to constant error which would not affect the correlations. The magnitude of the random errors constitutes the significant unknown. It is believed that these are reduced to reasonable limits by the technique employed. A check on this is provided by such cases as Nos. 1, 22, 25, and 50, where supplementary evidence for extent of the lesion in each case is available. The lesion in Case 1 has been reconstructed histologically by means of serial sections (193). The extent of the lesion, as based upon the sections, proved to be 3.72 per cent of the total cerebrum. The lesion as calculated from the chart of Case 1 was found to be 3.38 per cent.

[2] Drawn to approximate life-size (in the studio of the medical illustrator, Mr. Tom Jones) from the hemisphere studied in great detail by Economo and Koskinas (119).

Case 22 had complete removal of the left occipital lobe. All of the striate cortex was found to be contained in the surgical specimen, which was removed in one piece and studied histologically (206). The lesion, as calculated from the chart of Case 22, comprised 11.23 per cent of the cerebrum. The average percentage of the cortex of the total cerebrum represented by the occipital lobes, as given by Addison and Donaldson (3) for the normal brain is 9.26. The reader is referred to Bonin (45, 46) for a discussion of the limitations of absolute measurements reported in the literature.

Case 25 had a nearly complete removal of the right occipital lobe (206). While all the striate cortex was thought to be contained in the surgical specimen, the degree of extirpation was known to be slightly less in Case 25 than in Case 22. The calculated lesion for Case 25 is 10.01 per cent of the cerebrum.

TABLE 13

CORRELATIONS BETWEEN PERCENTAGE OF VISUALIZED CORTICAL BRAIN LESION AND IMPAIRMENT INDEX

Brain Lesion in Percentage	Pearson r*
Total lesion for all cases	−.091
Total lesion for frontal-lobe cases	+.090
Total lesion for nonfrontal-lobe cases	+.162
Lesion on superior surface of brain	+.096
Lesion on lateral surface of brain	−.291
Lesion on ventral surface of brain	−.156
Lesion on medial surface of brain	−.116

* None of these correlations is reliably different from zero.

Case 50 had a nearly complete removal or destruction of both prefrontal lobes. According to measurements made in this laboratory of Berry's dioptographic tracing of a normal brain (35), these structures include 31.68 per cent of the cerebrum. The calculated lesion for Case 50, known to be somewhat less than total removal, is 28.27 per cent. There thus appears to be a reasonably close agreement between our lesions as charted and external checks. Our method of charting would seem to justify the use of correlation technique.

Another possible source of a spuriously low correlation between degree of impairment and extent of lesion lies in the nature of our impairment index. The index is an over-all measure. If it were invalid for reflecting functions represented in the brain, we would expect only a chance correspondence with the extent of brain lesions. We have seen, however, that our impairment index does, in fact, reflect brain functions and, further, that these functions appear to have a maximal representation in the frontal lobes. The latter fact is compatible with a regional-localization

view of the cortex and with the general lack of relation between degree of impairment and extent of the lesions for the cortex as a whole.

Mass-action hypothesis.—Lashley (286) has reported a correlation coefficient of 0.86 between errors for initial learning of an eight cul-de-sac maze and the extent of cortical damage in rats, measured in percentage of the total cerebrum. For postoperative retention of this variable and size of lesion he obtained a coefficient of 0.59. Comparable data have been lacking for man. The relatively high magnitude of these correlations, and of others obtained for deterioration and damage within each of the cytoarchitectural fields of the rodent cortex, has constituted the major challenge to the localization hypothesis of the present century: "We have seen from the data presented in the preceding section that retardation is produced by a lesion in any of the cortical fields and that diverse lesions of equal magnitude produce, on the average, equal effects. The approximately equal correlations within the single (cycoarchitectural) fields further show that the retardation is dependent solely upon the extent of destruction, irrespective of its locus within the cerebral hemispheres" (286, p. 68).

How are we to account for this discrepancy between Lashley's findings for the rat and our findings for man? Several possibilities must be considered:

A. The human brain is structurally highly differentiated in comparison with the rat brain (145). The latter has a smooth, unconvoluted cortical surface, whereas the former is highly convoluted and differentiated into cytoarchitectural fields. More than one hundred areas of distinctive cellular structure have been identified in the cortex of the human brain (cf. Economo and Koskinas [119]) in contrast with less than ten such areas in the rat (145).

B. There is some reason to believe that a general process of encephalization of functions has paralleled the phylogenetic evolution of the human brain (310). Functions which are less dependent upon the integrity of the cortex in the rat have become more highly differentiated and, hence, more corticalized in man. This principle is perhaps most apparent, as yet, in the evolution of the motor areas of the brain (68) but may ultimately be shown to apply in the corticalization of speech mechanisms and in the sensory fields.

C. It is quite possible that the behavioral functions tested by Lashley in the rat and by the writer in man are not homologous functions. As pointed out in an earlier chapter, Lashley was forced, for methodological reasons, to equate learning mechanisms with the mechanism of intelligence, whereas we have found it necessary to distinguish between these

functions. The writer's methods for man secure a single, "naïve" sample of adaptive behavior. The influence of learning, although not entirely eliminated, is thus kept to a practical minimum. In contrast, Lashley's use of the maze situation as an indicator placed the emphasis at the outset upon an experimentally learned situation for the rat. We may concede a relation between learning and intelligence without conceding an identity of these processes. We have earlier noted that the nature of the deterioration found by Lashley may possibly reflect an alteration in our P or power factor. In this connection, it is probably significant that the P factor, as it emerges from our data, appears to be diffusely represented in the cortex even though it seems to be especially related to the prefrontal cortex. It may thus reflect the "central dynamic state" tentatively postulated by Lashley as the cortical representation of Spearman's general factor or G.

D. The regional-specialization view of biological intelligence, as supported by our data, is not necessarily incompatible with the principle of mass action postulated by Lashley if we make allowances for the factor of encephalization of function. Our findings indicate that not all cytoarchitectural boundaries establish functional limits on the localization of our basic factors. While the best performances of our temporal-, parietal-, and occipital-lobe cases bear this out, the number here is too small to justify correlation technique. Lesions in any of these anatomical zones do not necessarily produce the degree of impairment found in every one of our frontal-lobe cases. The zero correlation obtained for all types of cases between impairment and extent of lesion indicates that the principle of mass action does not apply for the human cortex as a whole for all our basic factors. Likewise, the zero correlations for our frontal-lobe and non-frontal-lobe cases indicate that the principle does not hold for such gross anatomical subdivisions of the brain. Our data do not yield evidence on the question of finer subdivisions within the brain—for example, within particular cytoarchitectural fields. The lesions in our human cases are not experimental lesions. They occurred primarily as accidents of nature, subsequently elaborated by the neurosurgeon without detailed reference to cytoarchitectural boundaries. Only when we can direct our analysis to the details within particular cytoarchitectural fields can we ascertain whether the principle of mass action has been retained at all in the process of encephalization of biological intelligence within the human cortex.[3] The facts of behavior, some of which are noted in chapter xvi, make it very improbable that nature has discarded this important principle.

[3] It is possible that, with gyrectomies now being performed in some neurological centers this occasion is not far distant.

CHAPTER XV

IMPAIRMENT INDEX AND CLOSED-HEAD (BRAIN) INJURIES

OUR data permit a further line of analysis which bears upon the validity of the basic factors in biological intelligence. Included in our group of patients for whom an impairment index has been determined are a substantial number of individuals convalescing from the effects of a traumatic closed-head (brain) injury.

Through co-operation with Gardiner General Hospital, an army hospital located in Chicago, a considerable number of closed-head-injury cases came to the attention of the writer throughout the recent war.[1]

Little is known concerning the essential mechanism in the production of the posttraumatic syndrome. It is well recognized that following mild or severe blows to the head, with or without an associated interval of unconsciousness, the individual may show complete signs of recovery (128, 193, 357, 390, 416). It is also well recognized that even a relatively mild trauma to the head may be followed by alterations in ego functions which impair the social capacities of the individual permanently (117, 144, 176, 184, 193, 344, 358, 388, 390, 416). But even in such instances it is frequently difficult or impossible to demonstrate the presence of actual brain damage ante mortem by existing methods. Clinical neurological examinations, formal psychometric studies, pneumoencephalography, electroencephalography, and clinical biochemical tests may yield consistently negative findings following the period of acute trauma. This fact has given rise to a growing opinion that the essential factor in the posttraumatic syndrome is the premorbid personality structure of the individual (128, 193, 249, 416). This view is commonly supported by the observation that a delay interval of weeks or months may transpire between the time of injury and the onset of behavioral abnormalities. Yet, it is well known that in the case of many brain tumors, a delay interval of months or years may precede the onset of behavioral symptoms in the patient (8). It is further known that many

[1] The writer is indebted to the commanding officer, Colonel John R. Hall; Colonel William Denny and his successor as chief of medicine, Colonel Paul Shallenberger; and Lieutenant Colonel Parks W. Richardson and Captain Charles O. Sturdevant, chiefs of neuropsychiatry, for their courtesy in referring cases for study.

brain tumors, sufficiently advanced in size to produce space-occupying lesions, may remain behaviorally "silent" throughout the life-span of the individual. We may refer once again to the study of Grünthal (184), who followed a small group of posttraumatic head cases over a period of several years. These cases acquired elaborate medical histories, at the hands of many medical specialists, ranging from diagnoses of functional neurosis to malingering and compensation neurosis. In no instance was a diagnosis of brain injury made. Yet, of sixteen cases in which it became possible to make a histological analysis of the brains, twelve had extensive damage in the frontal lobes attributable to the traumatic episode. In four cases the histopathology was ambiguous.

We may examine here the results which were obtained for one hundred and forty-seven cases of traumatic head injury associated with a variable interval of unconsciousness and with variable degrees of invalidism. All these cases had been hospitalized for an acute or chronic head injury. They were all ambulatory at testing and were examined individually in the writer's laboratory. We shall dispense with the clinical histories of these cases since they do not provide crucially validating information on the extent of impairment of brain functions. Short abstracts of these histories along with the impairment index found for each case will be published elsewhere.

In Figure 17 is shown the distribution of our impairment index for the one hundred and forty-seven closed-head-injury cases in the unshaded portion of the histogram. As reference groups, the impairment index for our thirty control subjects (shaded gray) and for twenty-four cases of either unilateral or bilateral frontal lobectomy (black) are also presented. It may be noted that 70 per cent of the closed-head-injury cases have an impairment index of 0.4 or above and 55 per cent an index of 0.5 or above, while 9 per cent have an index of 0.9.

Since we have found that the functions reflected by our impairment index are maximally represented in the frontal lobes of the brain, does it follow that the closed-head-injury cases with an impairment index falling in the range of our neurosurgical frontal-lobe cases have frontal-lobe damage? It is believed that the probabilities are relatively high that such is the case.

Is there any mechanical reason why a blow to the head should tend to produce damage in the frontal lobes more commonly than elsewhere in the brain? If so, our finding of frontal-lobe "signs" in a substantial proportion of our closed-head-injury cases would be in line with expectancy and with our quantitative findings for neurosurgical cases.

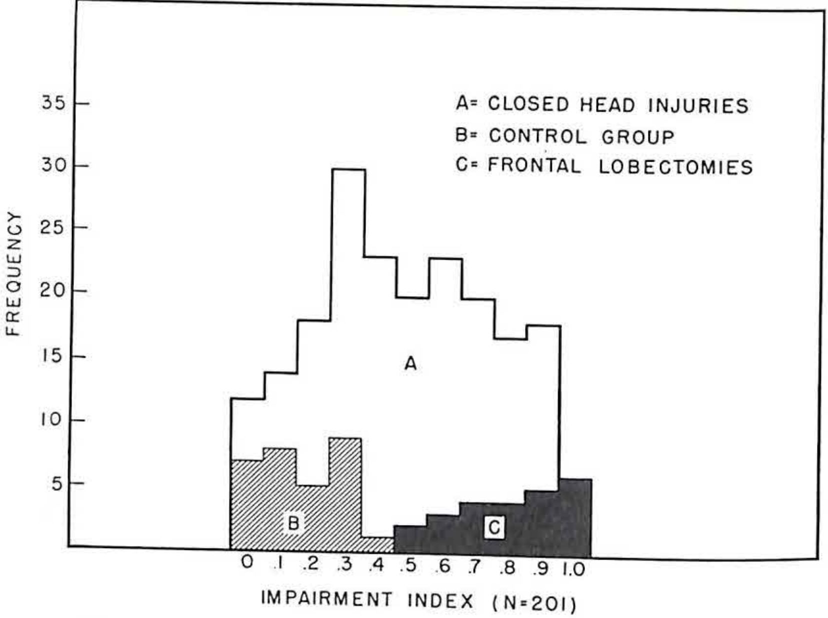


FIG. 17.—Distribution of Halstead impairment index in *A*, closed-head-injury patients; *B*, control group; and *C*, frontal lobectomies. Note that a considerable number of the cases in *A* have impairment-index scores similar to frontal lobectomies in *C*.

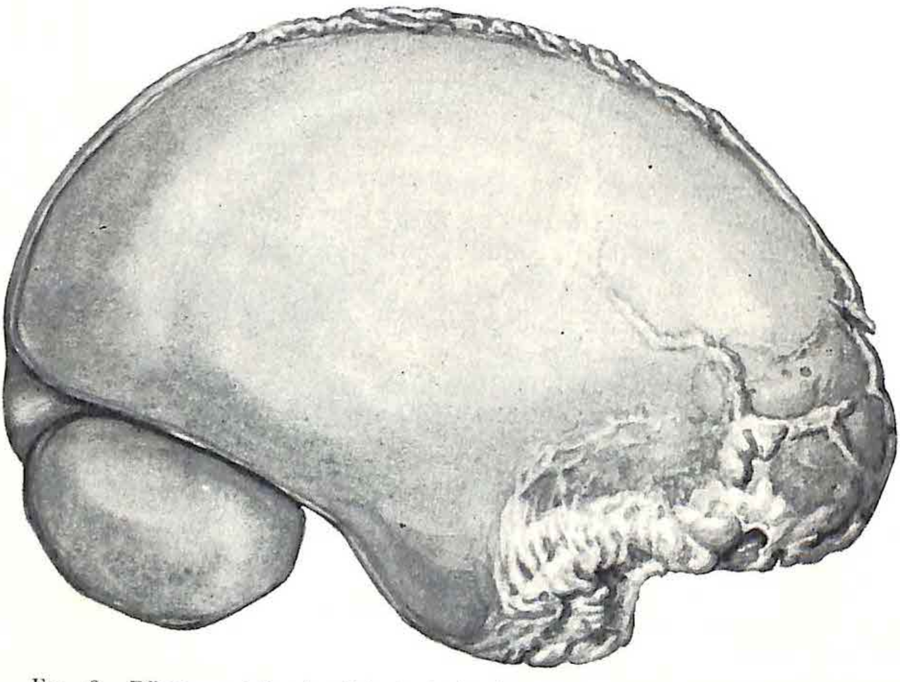


FIG. 18.—Effect on a jelly of a violent rotational jerking in its plane. Note that maximum tearing of the jelly mass occurs at the orbital surface of the frontal lobe and at the tip of the temporal lobe. (Reproduced from A. H. S. Holbourn [233] with permission of the publishers of the Lancet.)

Holbourn (233), an English physicist, has recently analyzed the shearing strains and stresses induced in the brain by a blow to the skull. Using the methods of photoelasticity and gelatin models of the brain, he has supplemented a theoretical analysis with empirical demonstrations that a concussive blow inducing rotational shear to any part of the skull tends to produce selective damage to the cortex of the frontal lobes. The middle and hind parts of the brain are apparently well damped to blows while the frontal poles shear across the sphenoidal ridges, tearing the meninges and brain substance and inducing hemorrhage. A retouched photograph of

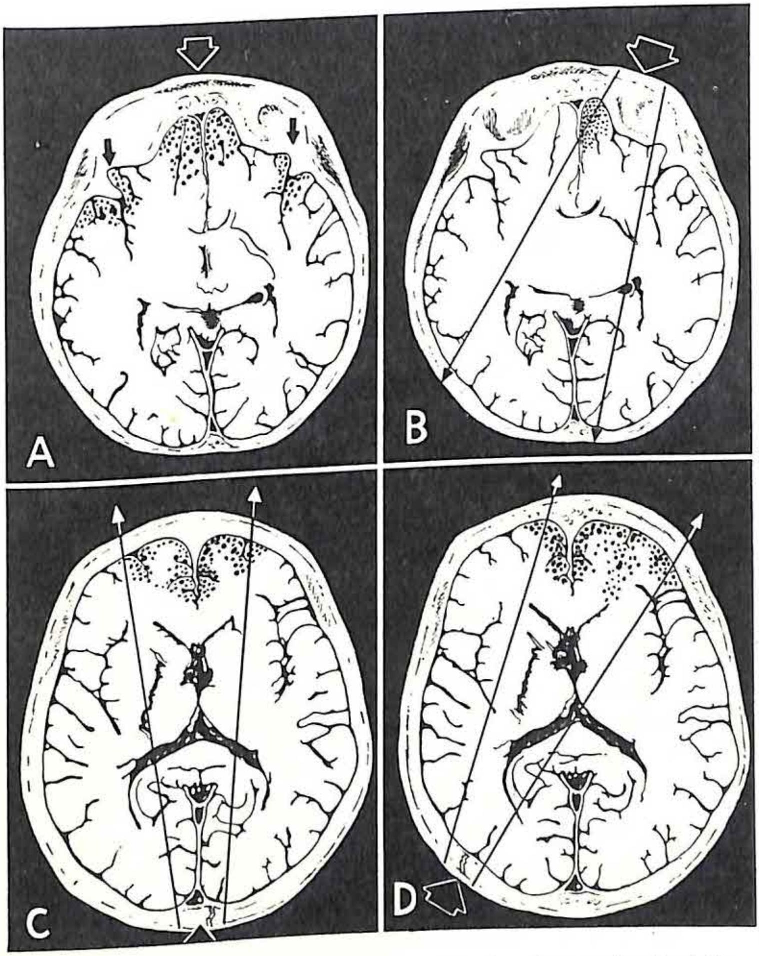


FIG. 19.—Line drawing of autopsy specimens of brains showing localized hemorrhage (shown as stippling) in the prefrontal lobes resulting from fatal trauma to various regions of the skull (point of impact indicated by large arrow). (Reproduced from C. B. Courville [89] with permission of the publishers of Archives of Surgery.)

one of Holbourn's experimental preparations showing this selective effect is reproduced in Figure 18 from the original publication.

Courville (89) recently analyzed autopsy findings on the brains of individuals following fatal head injuries and reached the same conclusion as Holbourn (see Fig. 19).

This evidence thus provides independent support for our quantitative results for the impairment index in head-injury and in neurosurgical patients. It likewise bears directly on the question raised in chapter xiv concerning the impairment index and localization of function. The impairment index appears to be a good reflector of frontal-lobe deficit and to provide a promising tool for evaluating this component in cases of mixed psychopathy, as in the posttraumatic syndrome.

CHAPTER XVI

BRAIN FUNCTION AND THE PROBLEM OF EQUIVALENCE

THE problem of sensory and motor equivalence in behavior has been emphasized by Lashley (286), Klüver (271), and Halstead (191). Stated in simple terms, the problem arises from the fact that the degrees of freedom for variance in behavior must be exactly paralleled in the functional organization of the brain. Lashley's original statement of the problem gave rise to much controversy and misunderstanding on the part of those who failed to see in it an alternative, no less deterministic, view of stimulus-response coupling (228, 333).

We have considered some of Lashley's evidence, based upon maze learning, which led him to the conclusion of mass action. Impairment of maze behavior was found to be proportionate to the cortical lesion regardless of location (286). Lashley's basic findings have subsequently been confirmed by Loucks (299), Maier (304, 305), Lashley (287), Lashley and Wiley (291), and others. A similar principle was found to hold within some areas of the bird cerebellum by Halstead (188, 208) for alteration in the direction of extinction of a labyrinthine reflex, a process recently shown by Yacorzynski (435) to be somewhat less markedly under cerebral control. For discrimination habits directed at particular cytoarchitectural areas the results obtained by Lashley were equally striking. For the visual (striate) area, for example, where there is evidence available for a virtual point-to-point projection of the retina onto the cortical surface, no fragmentation or disturbance of a simple light-dark discrimination habit occurred until more than half of the cortical region was destroyed. Memory for simple visual patterns, such as circles, squares, triangles, was not disturbed by destruction of up to 90 per cent of the striate areas, exclusive of the binocular projection. No part of the destroyed areas proved to be more critical than others. All parts were thus equipotential. It seems that a similar result is yielded by conditioning to tones (cf. Girden [167]). Total destruction of the auditory areas in an animal conditioned to a particular tone abolishes the response, but the response is unimpaired if any small part of the area remains intact.

Subcortical lesions placed by a stereotaxic instrument have been found to be effective for some habits, but it is not yet clear that the essential mechanism of impairment here is the same as for the cortex (60–63).

While emphasizing the holistic role of the cortex in certain forms of adaptive behavior, Lashley has never renounced the principle of regional localization for some habits. Thus, his visual discrimination tasks were found to be undisturbed by cortical lesions anywhere outside the striate area (286). On the other hand, his holistic conception could scarcely predict the high degree of localization in the frontal lobes of the rat of certain high-order habits involving "reasoning," "expectancy," and "delayed alternation" as has been found by Stellar, Morgan, and Yarosh (382) and by Morgan and Wood (325), with supporting evidence from Epstein and Morgan (122) and Loucks (299). As early as 1907, Franz (150) had trained cats and monkeys on Thorndike puzzle boxes and examined their behavior after he had induced lesions in the brain. He concluded that, when the frontal lobes are destroyed, such recently formed habits are lost. Comparable lesions elsewhere in the brain did not disturb such habits. He observed that unilateral lesions of the frontal areas did not produce loss of recently formed habits.

Jacobsen (241–43) reported in 1936 a series of experiments on monkeys designed to explore further the leads opened up by Franz. He employed five problem-box situations, roughly graded as to difficulty, and a simple pattern-discrimination test. He further utilized a delayed-response situation wherein the animal was required to choose one of two cups, loaded with food lure in its presence, following a delay interval in which the cups were hidden from the animal's view. Some of Jacobsen's results proved to be essentially the same as those of Franz (150). Unilateral lesions caused no impairment of performance on any tests. There was no evidence of hemispheral dominance.

Bilateral lesions of the frontal association areas, but not of other parts of the brain, did impair performance on the delayed-response situation. Total destruction of the frontal areas "completely and permanently abolished the habit," whereas subtotal lesions caused a shortening of the interval during which memory was effective. (Whether the impairment here is directly proportionate to the lesion—mass-action principle—is not brought out in Jacobsen's small number of cases.) It is of interest, in view of Franz's opposite conclusion, that bilateral frontal lesions did not abolish memory for the simple problem-box habits or for the visual-discrimination habits.

Breslaw, Barrera, and Warden (52) found that removal of the post-

central convolution of the monkey did not impair retention of the delayed-response habit.

Finan (129) reported confirmation of Jacobsen's findings for the delayed-response situation. In addition to this indicator, he employed a shuttle-box habit and a temporal-discrimination maze habit in four mangabey monkeys. Unilateral lesions of the frontal areas were found to cause no impairment on any of the tests. Bilateral lesions of the frontal areas, while sparing the shuttle-box and temporal-discrimination maze habits, "totally and permanently" abolished the capacity to respond correctly in the delayed-response test.

Malmo (308), in a carefully executed study, employed moderately intense (3.7 millilamberts) visual stimuli in accenting right-left choices in a delayed-response situation with one macaque monkey and one mangabey monkey. The animals were trained under two conditions. Under condition 1, the accenting light before one of two food boxes was turned on for two seconds; the animal was retained in darkness for ten seconds and then permitted to choose for food. Under condition 2, following exposure of the accenting light for two seconds, the animal was retained in intermittent darkness in the sequence: D, 2.5 seconds—L, 5 seconds—D, 2.5 seconds, before critical choice. Following its training, both prefrontal areas were removed (back to area 8) in the mangabey. Postoperative tests revealed retention of the delayed-response habit under condition 1 but not under condition 2. The macaque monkey was tested following bilateral frontal lobectomy. This animal made chance scores under condition 2 but equaled the preoperative level of performance of the mangabey on condition 1.

Malmo was seeking the answer to the questions: (*a*) whether elimination of visual stimulation during the delay period would enable the frontal-lobe animal to perform successfully in a delayed-response situation, and (*b*) whether interpolated visual stimulation would affect such performance adversely in the frontal-lobectomized animal. His quantitative results seem to supply an affirmative answer to both these questions. His interpretation that the disturbance in memory for the tests is attributable to retroactive inhibition is open to serious question on methodological grounds. It seems probable that the optical situation for condition 1 in his experiments was adequate if not ideal for the production of persistent after images in his animals. It is this situation which produced the evidence for successful performance of delayed responses in his operated animals. (I have not found any evidence of abnormal phenomena of afterimage formation in human cases with frontal-lobe lesions.) On the other hand, the optical situation in condition 2 of Malmo's experiments would favor

extinction of persistent afterimages. It was under this situation that evidence for successful performance failed to appear in the operated animals. The fact that the mangabey was able to perform the test preoperatively under condition 2 does not, *ipso facto*, specify the basis of its performance as being the same as for condition 1. In connection with the above comments, it should be noted that for optically induced forms of behavior the difficulties in specifying the level at which "seeing" comes off have been emphasized by Von Monakow (318), Poppelreuter (344), Klüver (270, 274, 275), Lashley (287, 288, 290), and Halstead (191, 193, 206).

Mass action versus localization.—It is apparent that, whereas evidence for mass action without specific localization has emerged for some experimental tasks for the rat (288), other evidence has emerged for specialization, of functions tapped by certain tasks, in the frontal lobes of the rat (122, 299, 325, 382). Except for the rat, the question of possible mass action within cytoarchitectural areas or within areas found to be critical for particular tasks has not been systematically explored. In addition to the rat, some degree of localization in the frontal lobes for particular tasks has been demonstrated for cat (150), monkey (129, 241, 242, 244), chimpanzee (245), and in our present results for man. We cannot yet compare results for the same "tasks" for different species, although possibly homologous visual functions having to do with form vision have been explored. Thus, any generalized principle of encephalization—shifting of functions from lower to higher centers progressively in the phylogenetic series—must rest for the present on meager evidence. The significant fact would seem to be that nature has employed two basic principles: mass action with equipotentiality for some functions and regional-localization specialization for others. Any satisfactory general theory of cortical organization must take both principles into account.

Evidence from man.—Except for the present findings, there are no comparable data available for man. General statements are to be found in the literature to the effect that the degree of impairment of mentality is related to the extent of the lesion. But impressionistic observations can add little to our understanding of the basic problems.

Quantitative observations on human cerebral lobectomies are not numerous. But where such data have been reported, they point to the fact that formal psychometric intelligence, as based on the Stanford-Binet test, may not be altered by removal of both prefrontal lobes or by unilateral removal of any other lobe of the brain (see German and Fox [165], Ackerly [2], Penfield and Evans [339], Brickner [53], Rowe [351], Halstead [190, 191], Lidz [293], and Hebb and Penfield [220]). Hebb (219) has

accumulated the most convincing array of evidence bearing upon this negative finding. For thirty-eight cases, drawn from the literature or from his own experience, the mean postoperative Stanford-Binet I.Q. was found to be 108 with a range from 54 to 152. For nineteen cases tested both pre- and postoperatively the mean drop in I.Q. was one point, with a range of minus fourteen points to plus eleven points. Of the nineteen cases, seven showed a loss, six no change, and six a gain after operation.

We have earlier reviewed the basic hypotheses which went into the evolution of the old and revised forms of the Stanford-Binet test. When faced with the patent absurdities presented by the average or even high I.Q.'s obtained in such cases as those of Ackerly (2), Brickner (53), Fox and German (148), and Nichols and Hunt (330), where the usable intelligence of the individuals is grossly impaired, one wonders in what sense the term "intelligence" can properly be applied to this test. It is of interest that Ackerly's case with bilateral frontal damage is Case 50 in our series (see Appendixes A and B). When examined in this laboratory in 1945, this patient, whose Stanford-Binet I.Q. is 106, was found to have an impairment index of 0.9 on our scale, which ranges from 0.0 to 1.0.

If we examine the content of the Stanford-Binet test from the standpoint of our basic factors (C, P, A, D), we find that its content makes substantial demands on C (central integrative field factor), much less demand on A (factor of abstraction) and P (power factor), and a substantial demand on our D factor (specific abilities).[1] Since these specific abilities are generally distributed throughout the population—probably in a Gaussian fashion—any scale which gives an over-all measure of them will similarly show a Gaussian distribution—the normal distribution curve.[2] The fact that the scale shows a moderately high correlation with ability to do secondary-school work, for example, validates neither the scale as a measure of intelligence nor, for that matter, the school work commonly selected as worthy tasks. One could probably arrange the optical or temperature components, for example, of school environments in such a way that quite different indicators would become "valid" in predicting performance—if, indeed, this has not already been demonstrated. Whether biological intelligence is a critical factor at all in successful performance of school work or in setting the tasks for it remains to be demonstrated. Certainly there is some reason to suspect that the school situation may

[1] We found no change in I.Q. in alternate forms of the revised Stanford-Binet in three subjects exposed to experimental anoxia of sufficiently high grade to produce signs of impairment of biological intelligence (see chap. xiii).

[2] It would not be surprising if the extremes of the distribution for psychometric intelligence were found to be correlated with biological intelligence as defined here.

provide a mass medium for impressing neurotic modes of adaptation—the polar extreme of biological intelligence—upon the general population. But let us return to the technical subject of this chapter: brain function and the problem of equivalence.

Neural equivalence.—While it has not yet been possible to test Lashley's hypothesis of equipotentiality of neural elements within cytoarchitectural fields for man, findings have been made which bear upon the basic problem. Much of the structural design of the projection of the retina onto the cerebral cortex has been worked out. In general, it indicates a point-to-point projection (59), but many of the essential details for central or macular vision are lacking. This lack is a serious one in view of the great importance of central vision in our daily lives. Evidence available from the detailed comparative anatomical studies of Minkowski (316), Brouwer and Zeeman (59), Clark (84), Walker (412), and others points to a unilateral projection of each half of the macula, divided along the vertical axis, onto the cortex of the contralateral occipital lobe. Removal of one occipital lobe thus produces a splitting of the macular visual field with associated hemianopia. The evidence for this conception in infrahuman animals is histological in character since satisfactory methods for mapping the details of the visual field in such forms have not yet been developed. Two cases from our present series (Nos. 22 and 25) have enabled us to explore this relation in man in great detail. In Case 22 the entire left occipital lobe was removed; in Case 25 virtually the entire right occipital lobe was removed. In each instance the operative specimen has been studied histologically to verify the extent of the ablation. For the details of this analysis as well as the methods employed in controlled mapping of the central and peripheral visual fields, the reader is referred to the original publication by Halstead, Walker, and Bucy (206).

The striking feature in these two cases is that the evidence yielded for cortical representation of macular vision is contradictory. In Case 22 the evidence indicates sparing of macular vision and, hence, bilateral projection of the macula. In Case 25 the evidence, equally good, indicates splitting of macular vision—in line with the findings for infrahuman forms—and, hence, unilateral representation of macular vision in the striate area. In Case 22 preoperative visual acuity (Snellen) was found to be: right eye, 0.8 + 3; left eye, 1.2 − 2; as compared with postoperative findings of: right eye, 0.6 − 2; left eye, 0.8. The amount of reduction in visual acuity for each eye, about 0.2, is the same in both cases. Yet the neural coupling in the two cases was found to be very different. In this fundamental visual function, neural equivalence in the type of projection of

structural elements has been demonstrated. There is no test by which it can be determined whether any given normal individual has unilateral or bilateral projection of his maculae. Nor can one design be shown to be superior to the other in mediating central vision. In the writer's experience with additional cases, fifty-fifty frequency is strongly suggested. It is probable that the macular coupling in any given individual hinges upon an embryological accident (419).

Pattern theory of neural equivalence.—Lashley (288) has criticized the various theories for neural equivalence offered by McDougall, Pavlov, Koehler, himself, and others. Writing in 1942, he proposed a pattern theory of neural equivalence. He assumes that the integrative functions of the cortex are carried out by transmission over short intranuncial fibers within the cortex since long connecting fibers have, in general, not been found. He cites the anatomical studies of Lorente de Nó (298) in support of the notion of reduplication of cortical association elements. Excitation started at any point in the cortex spreads from that point throughout the system by means of reverberating circuits. With several or many points of excitation, interference patterns will be formed in a way "somewhat analogous to the transmission of waves on the surface of a fluid medium." The interference pattern of excitation, upon spreading to a motor point, thus becomes the vehicle of propagation of the memory trace.

Since the points in the cortex are reduplicated or equipotential in Lashley's scheme, there can be no preferred direction of transmission of the initial excitation and, hence, of the interference patterns. Transmission to the motor points would presumably be along the shortest path, that is, from the sensory fields directly to the motor cortex. Provision for intellect in the system is made by virtue of the fact that "the receptive areas of the cortex are themselves capable of processes of generalization not fundamentally different from 'higher' intellectual activities" (288, p. 304).

Lashley's holistic theory accounts well for the sparing of many forms of behavior following cerebral damage, including the human-like appearance of individuals in whom both prefrontal lobes have been removed. Hebb, his student, has contributed valuable support to the theory by presenting convincing evidence that removal of the prefrontal lobes does not impair psychometric intelligence. We have found and described the structures of a biological intelligence which is different from psychometric intelligence. We have found further that this biological intelligence not only is represented in the brain but is maximally represented in the frontal lobes of the brain and, more specifically, in the cortex of the frontal lobes. Since

Lashley's theory makes no provision for such localization or specialization, it can provide no basis for interpreting our results.

A gradient theory of neural equivalence.—The function of any theory is to account for known facts and to open up rational avenues for research. The facts which a theory of neural equivalence must take into account include those of levels of consciousness, volitional control of behavior, sensory and motor equivalence, possibly some forms of affective equivalence, in addition to those facts pointing to localization or specialization of some functions in the brain.

The impressive growth of the field of electronics in recent years finds this writer envious of the insight which has been gained into fundamental mechanisms and relations. Analogies and homologies drawn from this field appear useful to students of brain physiology. Grossly, perhaps the most conspicuous feature of the brain is its apparent symmetry. This basic symmetry, plus the bilateral representation of the distance-receptor fields in the two hemispheres, suggests the analogy of a high-fidelity electronic amplifier (327). This amplifier is characterized by a large undistorted power factor and a remarkable sensitivity. From a technical standpoint, nature seems to have designed this amplifier with a balanced push-pull type of input (e.g., crossed projection of optic and auditory nerves) and balanced intermediate stages of amplification as well as output points of discharge. For further regulation of the system the principle of inverse feedback (diversion of part of the amplified energy for regulation of the input) was introduced. To adjust the system to a wide range of input loads, each stage of amplification is connected in parallel with the one ahead and with the output stages (an interesting arrangement as yet untried by man).

Around this instrument has been placed a structure (the outer layers of the cortex) in many ways not unlike the cathode-ray screen and focusing circuits of an electronic oscilloscope. Onto its screen are projected bursts or waves of excitation which, when brought under regulation by appropriate sweep circuits, emerge as unclosed Lissajous-like figures or patterns of excitation. A powerful polarized D.C. potential, possibly like the corneoretinal potential (189, 315, 326), gives forward impetus to these excitation figures, carrying them up to the frontal poles of the brain (P factor). As they sweep across the cortex, local focusing circuits (reverberating) contribute and maintain detailed focus or identity. At the frontal lobes the open extremes of the figures meet and closure or completion occurs (high-level consciousness). In closed form the figures become adequate stimuli to the mosaic of the motor cortex. Closure or completion of

the figures may occur before the prefrontal lobes are reached, with an associated lower degree of consciousness. Thus, the individual without prefrontal lobes will retain remarkable similarity to the normal in many respects but will be significantly different in others.

It is possible that strong electrotonic cortical fields on the ventral surfaces of the brain (affective fields), normally bending the excitation figures toward completion or closure, are delayed in this by inverse feedback (inhibitory) regulation from the prefrontal lobes (374). When the regulating influence of these latter structures is disturbed by lesions, the whole system is, to some extent, unbalanced. Closure of the neural image is premature and gives rise to a "driven" character in the performances of individuals with such lesions. For biological intelligence, in brief, has been substituted "biological neurosis." Biological neurosis can probably be duplicated in every essential detail without the requirement of brain injury, at least in the present meaning of this term, through learning (habits).

Fanciful and speculative as this fragmentary sketch may appear, it nevertheless demands the serious attention of students of brain physiology, for example, whose searches for a neural-excitation image have thus far proved fruitless. It may be that the electrical currents involved in the propagated image do not rise above the electronic noise level of modern electrometers. On the other hand, the image might conceivably be carried as a standing-wave component of the electroencephalogram, where its presence, even if noted, might remain uninterpreted. Or it may be necessary to turn to chemistry for ultra-sensitive fluorescent or phosphorescent emulsions with critical time constants which, on films brought into close proximity with the cortex, might enable us to visualize underlying electrical events. Recently developed neurosurgical approaches to the problem of epilepsy (334, 337) in terms of locating trigger zones in the exposed human cortex may present favorable opportunities for explorations along the lines indicated above.

It is of interest in connection with our gradient theory of cortical excitation, that partial or complete sectioning of the corpus callosum, the major commissural connection between the two hemispheres of the brain, apparently results in neither general nor focal alteration in behavioral functions (370, 411).

The ego and the prefrontal lobes.—In recent years psychiatry has moved more and more in the direction of a conception of ego failure as the basis of mental disorder, and herein lies real hope for scientific advance. The present studies have revealed that at least some of the factors which are

essential for ego formation and growth have their maximal representation in the prefrontal lobes of the brain. Removal of these structures in man effectively reduces to the point of virtual elimination ego control of behavior. The personality of such individuals is de-differentiated to a stage at which once again orientation and security are dependent upon the world of senses. They are stimulus- or "sign"-bound in the control of their motilities. Reduction of their ability for abstraction (A factor) has limited the world of symbols (relations among relations) available to them. Reduction of the cerebral power factor (P) has further increased the extent to which their motilities are dominated by emotional or affective influences. As a result, they can no longer tolerate the psychologically "new" without undue anxiety. A principle of immediate effect prevails throughout. Valuative judgments toward long-range goals give way to the rewards or punishments of the tangible present. They manage to survive through progressive de-conditioning of their aggressivity but always in terms of a highly stereotyped mode of living. They have lost a biological heritage which attains its zenith in Homo sapiens, the capacity for ego growth and differentiation.

The psychiatrist may see in this discussion a prototype of the severely neurotic, chronically depressed, anxiety-ridden mental patient, and in the opinion of the writer he would not be in error in doing so; for our observations suggest that those individuals who habitually do not or cannot use their prefrontal lobes in controlling their motilities are scarcely less lacking in adaptive intelligence (ego strength) than those who no longer have these portions of the brain.

The problem of mental disorder is assuming alarming proportions in the modern world. Egos that might have been adequate for the simpler life of another time are failing in increasing numbers to be equal to the demands of modern living. Medicine has done much to lengthen the life-span of man. But it has accomplished this with bodies that have already been formed in terms of a species norm. It cannot restore a missing or withered limb, or an absent eye. Neither can it hope to restore an ego that has failed to develop or has been greatly altered by damage to the frontal lobes. If man is to secure rational control of his destiny, we must direct more attention and effort to the task of building healthy egos in our population. Only a seemingly endless study of the biological and social dimensions of organisms, including man, can hope to show us the way.

CHAPTER XVII

SUMMARY AND CONCLUSIONS

In Part I of this volume the general question of the structure of biological intelligence was raised. Psychometric, neurological, psychiatric, and psychoanalytic conceptions of intelligence were examined and found to yield confused and contradictory views. In seeking quantitative indices of biological intelligence, two hundred and thirty-seven subjects, including normal control individuals and patients with various types of brain lesions, were examined with a battery of twenty-seven neuropsychological tests, several of which were employed for the first time in this study. Factor analyses of those variables amenable to Pearson correlation technique, both in orthogonal and oblique solutions and in Thurstone's multiple-factor solution, yielded a four-factor description of performance on the tests. These factors were found to be valid for differentiating the effects of brain injury and have been made the basis of a four-factor theory of biological intelligence. The nature of these factors may be summarized as follows:

1. A central integrative field factor C. This factor represents the organized experience of the individual. It is the ground function of the "familiar" in terms of which the psychologically "new" is tested and incorporated. It is a region of coalescence of learning and adaptive intelligence. Some of its parameters are probably reflected in measurements of psychometric intelligence which yield an intelligence quotient.

2. A factor of abstraction A. This factor concerns a basic capacity to group to a criterion, as in the elaboration of categories, and involves the comprehension of essential similarities and differences. It is the fundamental growth principle of the ego.

3. A power factor P. This factor reflects the undistorted power factor of the brain. It operates to counterbalance or regulate the affective forces and thus frees the growth principle of the ego for further ego differentiation.

4. A directional factor D. This vector constitutes the medium through which the process factors, noted here, are exteriorized at any given moment. On the motor side it specifies the "final common pathway," while on the sensory side it specifies the avenue or modality of experience.

In Part II of this volume the extent to which the above factors are represented in the brain was tested. The best available measures for the different factors were combined in the form of an impairment index. When applied to normal control subjects and to patients with various types of brain lesions, the following results were obtained:

5. In comparison with other types of subjects, individuals with damage to the frontal lobes have high impairment-index scores. The mean impairment index for frontal lobectomies was found to be about six times that for normal control subjects and about three times that for nonfrontal lobectomies.

6. This relation holds whether the lesion is unilateral or bilateral and whether it is on the right side or on the left side of the brain.

7. There is no quantitative evidence of hemispheral dominance yielded by this study.

8. There is no relation between the degree of impairment and the extent of the lesion. The obtained correlations are not significantly different from zero.

9. This is true for the brain as a whole, for the cortical areas of the prefrontal lobes, and for cortical areas outside the frontal lobes.

10. No evidence for a general principle of mass action is yielded by this study. On the other hand, the available data do not bear upon mass action in restricted cytoarchitectural areas.

11. Bilateral subcortical lesions of the frontal lobes, as in lobotomies, do not disturb the functions reflected by the impairment index.

12. Experimental anoxia (of the brain) in the normal adult produces selective impairment and recovery of biological intelligence at a stage at which psychometric intelligence is unimpaired.

13. The impairment of biological intelligence in patients convalescing from recent closed-head injuries is similar to that in frontal lobectomies.

14. The impairment of functions reflected by the impairment index is independent of psychometric intelligence.

15. The impairment is independent of disturbances in language functions.

16. The impairment is not referable to any sensory or motor defects.

17. A review of the literature on cerebral function in mammals other than man indicates that, while some functions appear to be diffusely represented in the cortex, others are regionally localized in the brain.

From these facts the following inferences are drawn:

1. Biological intelligence is a basic function of the brain and is essential for many forms of adaptive behavior of the human organism. While it is

represented throughout the cerebral cortex, its representation is not equal throughout. It is distributed in a gradient with its maximal representation occurring in the cortex of the frontal lobes.

2. The nuclear structure of biological intelligence comprises four basic factors which, in unified fashion, enter into all cognitive activities. While these factors make possible the highest reaches of human intellect, their dysfunction, as produced by brain damage, may yield progressively maladaptive forms of behavior, or "biological neurosis."

3. The frontal lobes, long regarded as silent areas, are the portion of the brain most essential to biological intelligence. They are the organs of civilization—the basis of man's despair and of his hope for the future.

APPENDIXES

APPENDIX A

ABSTRACTS OF MEDICAL HISTORIES OF CEREBRAL LOBECTOMIES

The following group of neurosurgical patients was drawn for the most part from the neurological and neurosurgical services of the Albert Merritt Billings Memorial Hospital of the University of Chicago. Where patients have been referred to the writer for study by doctors at other institutions, this fact is noted in the summary of the history given below. Dr. Percival Bailey, Dr. Paul C. Bucy, or Dr. A. Earl Walker performed most of the operations, and all have co-operated fully in making the cases available for study.

Case

1. A 48-year-old American-born male; occupation: insurance broker. Entered University of Chicago Clinics (UCC) 9/4/35 with a history of severe headaches, occasional vomiting, blurred vision, and loss of weight. Operation performed 9/17/35 by Dr. Bailey involved partial extirpation of the left prefrontal lobe for removal of an olfactory groove meningioma. Recovery uneventful; patient discharged from hospital 9/28/35. Postoperative I.Q., 110 (Stanford-Binet). A detailed history of this patient has been published elsewhere (191, 193).
   Frontal lobectomy   Impairment index: 1.0
2. A 55-year-old American-born female of German descent; occupation: housewife. Entered UCC 2/1/31 with a history of pain in the back of the neck, past few months; headaches, last six months; loss of smell, several years ago; loss of memory recently; desire to sleep; constipation; difficulty in walking at times. Operation performed 2/13/31 by Dr. Bailey involved extirpation of meningioma of left olfactory groove. Recovery uneventful; patient discharged from hospital 3/3/31.
   Frontal lobectomy   Impairment index: 1.0
3. A 58-year-old Canadian-born female; occupation: none. Entered UCC 8/31/36 with a history of about ten major convulsions in eight hours. Operation performed 9/10/36 by Dr. Bucy involved left frontal osteoplastic craniotomy and partial extirpation of tumor and amputation of left prefrontal lobe. Recovery uneventful; patient discharged from hospital 10/6/36.
   Frontal lobectomy   Impairment index: incomplete
4. A 32-year-old American-born male of German descent; occupation: truck-driver. Entered UCC 12/3/37 with a history of convulsions since 1935. Operation performed 12/9/37 by Dr. Bucy involved left frontal osteoplastic exploration, partial extirpation of glioma. Patient aphasic for ten days postoperatively but recovered completely; patient discharged from hospital 12/21/37.
   Frontal lobectomy   Impairment index: 0.8
5. A 36-year-old American-born male; occupation: salesman. Entered UCC 6/23/37 with a history of generalized convulsions and progressive difficulty in speaking for the last seven years. Operation performed 6/29/37 by Dr. Bucy involved partial

extirpation of the left prefrontal lobe for removal of an astrocytoma. Recovery uneventful except for transitory motor aphasia; patient discharged from hospital 7/10/37.

Frontal lobectomy Impairment index: 0.7

6. A 51-year-old Irish-born male of Irish descent; occupation: police officer. Entered UCC 6/17/36 with a history of severe headaches, dizziness and unsteadiness on his feet for four months, and a loss of memory for some time longer. Operation performed 6/23/36 by Dr. Bucy involved a left frontal osteoplastic exploration and extirpation of glioma. Recovery uneventful; patient discharged from hospital 7/20/36. On 8/11/38 I.Q., 97 (Stanford-Binet, Form M).

Frontal lobectomy Impairment index: 1.0

7. A 53-year-old American-born male; occupation: machinist. Entered UCC 10/17/37 with a history of fainting spells since April, 1937, and mental changes. Operation performed 10/26/37 by Dr. Bucy involved amputation of left frontal lobe and partial extirpation of glioma. Recovery uneventful; patient discharged from hospital 11/7/37.

Frontal lobectomy Impairment index: 1.0

8. A 32-year-old Austrian-born male of Slavic descent; occupation: building laborer. Entered UCC 3/14/39 with a history of weakness, numbness in entire left side, duration nine months. Dizziness and headache (continual), duration nine months. Operation performed 3/19/39 by Dr. Bailey involved right frontal parietal region. Patient discharged from hospital 3/29/39.

Frontal lobectomy Impairment index: 0.9

9. A 47-year-old Lithuanian-born male of Lithuanian descent; occupation: journalist. Entered UCC 2/1/38 with a history of convulsions on the left side for approximately six years. Operation performed 2/8/38 by Dr. Bucy involved osteoplastic exploration and amputation of right prefrontal lobe. Recovery uneventful; patient discharged from hospital 3/2/38.

Frontal lobectomy Impairment index: 0.6

10. A 31-year-old American-born female; occupation: housewife. Entered UCC 7/24/39 with a history of repeated headaches. Operation performed 7/29/39 by Dr. Walker involved right prefrontal lobectomy. Recovery uneventful; patient discharged from hospital 8/15/39.

Frontal lobectomy Impairment index: 0.6

11. A 46-year-old American-born male; occupation: farmer. Entered UCC 5/3/37 with a history of headaches in right frontal region for one and one-half years. Six months before their onset he was butted by a ram from behind. His head struck a corncrib, and he was unconscious some minutes, but there was no further complication until one and one-half years ago. Operation performed 5/13/37 by Dr. Bailey involved extirpation of meningeal tumor in right frontal lobe and partial lobectomy. Recovery uneventful; patient discharged from hospital 6/12/37.

Frontal lobectomy Impairment index: 0.9

12. A 33-year-old American-born female; occupation: comptometer operator. Entered UCC 4/10/39 with a history of focal motor seizures since June, 1938. Operation performed 4/20/39 by Dr. Bucy involved osteoplastic exploration, decompression and extirpation of meningioma. Recovery uneventful; patient discharged from hospital 5/15/39. Right frontal lobe involved superficially.

Pressure on cortex of frontal lobe Impairment index: 0.5

13. A 19-year-old American-born male; occupation: farmer. Entered UCC 12/2/35 with a history of frequent headaches, progressive loss of memory, loss of ability to concentrate and loss of vision. Operation performed 12/5/35 by Dr. Bailey involved partial extirpation of left temporal lobe for partial removal of a glioma.

Recovery was uneventful except for persistent motor aphasia; patient discharged from hospital 12/15/35.

Temporal lobectomy Impairment index: 0.3

14. A 35-year-old American-born male; occupation: farmer. Came to the laboratory 8/22/38 after having been seen by Dr. Bailey. Ten years ago patient was completely paralyzed. Operation performed at another hospital involved the removal of a cyst from the left temporal lobe. For eighteen months following the operation the patient could not speak. Since then there has been progressive improvement from the paralysis and loss of speech.

Temporal lobectomy Impairment index: 0.3

15. A 33-year-old American-born female; occupation: housewife. Entered UCC 6/25/39 with a history of headaches. Operation performed 7/6/39 by Dr. Walker involved left osteoplastic craniotomy, partial extirpation of temporal glioblastoma, and subtemporal decompression. Recovery uneventful; patient discharged from hospital 7/11/39.

Temporal lobectomy Impairment index: 0.3

16. A 51-year-old German-born male; occupation: truck-driver. Entered UCC 7/7/36 with a history of generalized convulsions, strange tastes and odors during the last four months. Operation performed 7/11/36 by Dr. Bucy involved extirpation of the right temporal lobe for partial removal of an astrocytoma. Recovery uneventful; patient discharged from the hospital 8/3/36. Postoperative I.Q., 91 (Stanford-Binet).

Temporal lobectomy Impairment index: 0.3

17. A 36-year-old American-born male; occupation: none. Entered UCC 1/2/36. Two operations performed by Dr. Bailey involved right temporal craniotomy and partial extirpation of tumor. Recovery uneventful; patient discharged from hospital 2/13/36.

Temporal lobectomy Impairment index: 0.1

18. This case came to our attention through the courtesy of Dr. Harold Voris of Chicago. A 31-year-old American-born male; occupation: none. Entered Mercy Hospital 9/12/35 with a history of major and minor seizures for the last four and one-half years and severe frontal headaches during past year. Operation performed 9/13/35 by Dr. Voris involved partial extirpation of the right temporal lobe for removal of a "hard fibrous infiltrating tumor located 2 cm. below the surface. A cavity about 4×4×6 cm. was produced in the posterior part of the right temporal lobe." Recovery was uneventful except for residual focal seizures which could not be completely controlled with medication. Postoperative I.Q., 110 (Stanford-Binet). A second operation performed 2/4/39 involved partial extirpation of the left occipital and parietal lobes. Recovery uneventful except for left hemiparesis.

Temporoparietal lobectomy Impairment index: 0.4

19. A 19-year-old American-born male of Swedish descent; occupation: cement worker. Entered UCC 1/15/36 with a history of convulsions averaging three per week for six years and right-side weakness for six years. Operation performed 1/24/36 by Dr. Bucy involved extirpation of sclerotic cortex involving most of the left parietal cortex. Recovery uneventful; patient discharged from hospital 2/11/36.

Parietal lobectomy Impairment index: 0.1

20. A 28-year-old American-born female of Swedish descent; occupation: nurse. Entered UCC 5/17/37 with a history of four major convulsions in last year and loss of use of left arm and leg for two weeks. Operation performed 5/20/37 by Dr.

Bailey involved extirpation of right parietal glioma. Recovery uneventful; patient discharged from hospital 6/13/37.

Parietal lobectomy   Impairment index: 0.3

21. A 21-year-old American-born female of Italian descent; occupation: none. Entered UCC 7/13/36 with a history of Jacksonian attacks beginning in the left ankle, occurring every two days up to several times daily for approximately eleven years since the age of 9½. Operation performed 7/25/36 by Dr. Bucy involved extirpation of cerebral scar from left parietal lobe and osteoplastic exploration. Recovery uneventful; patient discharged from hospital 8/17/36.

Parietal decortication   Impairment index: 0.1

22. A 25-year-old American-born female of German descent; occupation: file clerk. Entered UCC 9/27/37 with a history of visual hallucinations for ten years and headaches and vomiting for eighteen months. Operation performed 10/23/37 by Dr. Bucy involved left occipital osteoplastic flap and amputation of left occipital lobe. Recovery uneventful; patient discharged from hospital 11/11/37. For a detailed study of the visual system of this patient see Halstead *et al.* (206).

Occipital lobectomy   Impairment index: 0.2

23. A 35-year-old American-born female of Danish descent; occupation: stenographer. Entered UCC 4/22/32 with a history of left occipital headaches and blurring of vision for four months. Operation performed 4/28/32 by Dr. Bailey involved intracapsular enucleation of meningioma of left occipital region. Recovery uneventful; patient discharged from hospital 7/6/32.

Occipital lobectomy   Impairment index: 0.0

24. A 22-year-old American-born female of Italian descent; occupation: housewife. Entered UCC 7/29/29 with a history of severe headaches for one month and blurred vision in the left eye for two or three weeks. Operation performed 8/2/29 by Dr. Bailey involved osteoplastic exploration and left occipital lobectomy. Patient discharged from hospital 8/26/29.

Occipital lobectomy   Impairment index: incomplete

25. A 27-year-old American-born female of German descent; occupation: secretary. Entered UCC 9/27/39 with a history of left hemianopia and headaches. Operation performed 10/10/39 by Dr. Bucy involved amputation of right occipital lobe. Recovery uneventful; patient discharged from hospital 11/3/39. For a detailed study of the visual system of this patient see Halstead *et al.* (206). Postoperative I.Q., 133 (Stanford-Binet, Form M).

Occipital lobectomy   Impairment index: 0.2

26. A 15-year-old American-born male; occupation: student. Entered UCC 10/4/38 with a history of frontal headache, three months; dizziness, five months; loss of balance, three months; pressure in ear, three months. Operation performed 10/6/38 by Dr. Bucy involved suboccipital craniectomy, evacuation of cyst, extirpation of tumor. Recovery uneventful; patient discharged from hospital 10/19/38. Postoperative I.Q., 109 (8/22/41—Wechsler-Bellevue).

Cerebellarectomy   Impairment index: 0.0

27. A 14-year-old American-born male; occupation: student. Entered UCC 9/20/39 with a history of headaches, blurred vision, vertigo. Operation performed 9/21/39 by Dr. Walker involved right frontal craniotomy, puncture of brain abscess, closure. Operation performed 9/27/39 by Dr. Walker involved evacuation and drainage of right frontal abscess. Recovery uneventful; patient discharged from hospital 11/25/39.

Patient re-entered UCC 3/5/44 with a history of convulsive attacks since October, 1940. Operation performed 3/9/44 by Dr. Walker involved resection of cerebral

scar, cranioplasty. Recovery uneventful; patient discharged from hospital 3/19/44.

Frontal lobectomy   Impairment index: 0.6

28. A 46-year-old American-born female; occupation: housewife. Entered UCC 1/5/42 with a history of fainting spells for six years. Operation performed 1/10/42 by Dr. Walker involved osteoplastic craniotomy, extirpation of right temporal meningioma. Recovery uneventful; patient discharged from hospital 1/21/42.

Re-entered UCC 1/9/44 with a skull defect resulting from operation here 1/10/42. Operation performed 1/15/44 by Dr. Walker involved cranioplasty. Recovery uneventful; patient discharged from hospital 1/26/44.

Temporal lobectomy   Impairment index: 0.0

29. A 46-year-old American-born female of German descent; occupation: housewife. Entered UCC 10/21/41 with a history of headaches for six years. Operation performed 10/23/41 by Dr. Walker involved left osteoplastic craniotomy, left frontal lobectomy. Recovery uneventful; patient discharged from hospital 12/4/41.

Frontal lobectomy   Impairment index: 0.9

30. A 63-year-old Polish-born male; occupation: gateman. Patient entered UCC 2/26/44 with a history of headaches for six weeks. Operation performed 3/11/44 by Dr. Walker involved craniectomy, right frontal lobectomy, removal of tumor. Recovery uneventful; patient discharged from hospital 3/19/44.

Frontal lobectomy   Impairment index: 0.5

31. A 35-year-old American-born female of Polish descent; occupation: housekeeper. Entered UCC 1/25/39 with a history of occipital headaches for over one year. Operation performed 1/28/39 by Dr. Walker involved left parietal occipital osteoplastic craniotomy, extirpation of intraventricular meningioma, decompression. Recovery uneventful; patient discharged from hospital 2/17/39.

Parieto-occipital lobectomy   Impairment index: incomplete

32. A 41-year-old Italian-born male of Italian descent; occupation: owner of grocery store. Entered UCC 9/2/40 with a history of convulsions for three years. Operation performed 9/17/40 by Dr. Walker involved osteoplastic craniotomy, left occipital lobectomy, biopsy of glioma of the pulvinar. Recovery uneventful; patient discharged from hospital 9/29/40.

Occipital lobectomy   Impairment index: 0.1

33. A 16-year-old American-born male of Polish descent; occupation: student. Entered UCC 7/2/39 with a history of headache, vomiting, fever, and coma. Operation performed 7/2/39 by Dr. Bucy involved right frontal craniectomy, evacuation of two brain abscesses. Operation performed 7/6/39 by Dr. Bucy involved extirpation of osteomyelitic bone, incision of dura mater. Operation performed 7/15/39 by Dr. Bucy involved evacuation and drainage of brain abscess, partial ostectomy. Operation performed 8/10/39 by Dr. Bucy involved multiple ostectomies and sequestrectomies, frontal bone. Recovery uneventful; patient discharged from hospital 9/5/39.

Re-entered UCC 11/10/39 with a history of occipital headaches, tenderness over right forehead, and swelling of right eyelid. Operation performed 11/11/39 by Drs. Walker and Bucy involved sequestrectomy, ostectomy of right supraorbital ridge. Recovery uneventful; patient discharged from hospital 11/20/39.

Re-entered UCC 8/14/41 to have postoperative defect in skull repaired. Operation performed 8/21/41 by Dr. Walker involved cranioplasty. Recovery uneventful; patient discharged from hospital 9/3/41.

Re-entered UCC 11/3/41 with a history of recurrence of sinusitis. Operation performed 11/4/41 by Dr. Walker involved craniotomy, incision and drainage of

brain abscess in right frontal lobe. Recovery uneventful; patient discharged from hospital 11/20/41.

Re-entered UCC 11/23/41 with a history of recurrence of sinusitis. Operation performed 12/4/41 by Dr. Walker involved removal of plate. Recovery uneventful; patient discharged from hospital 12/15/41.

Re-entered UCC 5/27/43 for above reason. Operation performed 5/29/43 by Dr. Walker involved cranioplasty. Recovery uneventful; patient discharged from hospital 6/8/43.

Frontal lobectomy Impairment index: 1.0

34. A 44-year-old American-born male; occupation: unemployed. Entered UCC 3/3/40 with a history of repeated attacks of faintness and feeling of unreality, three years; attacks of clonic contractions of neck muscles, three years; attacks of weakness of left arm and lack of co-ordination of both upper extremities, three years. Operation performed 3/7/40 by Dr. Walker involved right osteoplastic craniotomy, partial extirpation of deep-seated astrocytoma, intracranial decompression. Recovery uneventful; patient discharged from hospital 3/19/40.

Frontal lobectomy Impairment index: 0.8

35. A 29-year-old American-born female; occupation: clerk. Entered UCC 4/27/43 with a history of headaches, nausea, and vomiting for last fourteen months. Operation performed 4/29/43 by Dr. Walker involved removal of glioblastoma from left frontal lobe. Recovery uneventful; patient discharged from hospital 5/12/43.

Frontal lobectomy Impairment index: 0.6

36. A 49-year-old American-born female; occupation: none. Entered UCC 11/7/37 with a history of convulsions for one year. Operation performed 11/16/37 by Dr. Walker involved left frontal osteoplastic craniotomy, extirpation of meningioma. Recovery uneventful; patient discharged from hospital 11/28/37.

Frontal lobectomy Impairment index: 0.9

37. A 39-year-old American-born male of Scottish descent; occupation: laborer. Entered UCC 9/1/41 with a history of convulsions. Operation performed 9/9/41 by Dr. Walker involved osteoplastic craniotomy, partial left temporal lobectomy, and partial extirpation of astrocytomatous tumor. Recovery uneventful; patient discharged from hospital 10/4/41.

Re-entered UCC 3/12/43 with a history of vomiting of eight hours' duration, weakness for five months, and headaches from three to four months. Operation performed 3/16/43 by Dr. Walker involved secondary craniotomy, partial extirpation of recurrent left temporal astrocytoma. Recovery uneventful; patient discharged from hospital 3/30/43.

Temporal lobectomy Impairment index: 0.3

38. A 47-year-old American-born male of Norwegian descent; occupation: outside representative for telephone company. Entered UCC 3/6/45 with a history of fainting spells for five years. Operation performed 3/10/45 by Dr. Walker involved osteoplastic craniotomy, left temporal lobectomy, removal of meningioma. Recovery uneventful; patient discharged from the hospital 4/15/45.

Temporal lobectomy Impairment index: 0.9

39. A 26-year-old American-born female; occupation: housewife. Entered UCC 11/24/43 with a history of a "prickling feeling" left side of the face for nine months and headaches for one and one-half years. Operation performed 11/30/43 by Dr. Walker involved myoplastic craniotomy, subtotal resection of left temporal lobe, partial removal of astrocytoma. Recovery uneventful; patient discharged from hospital 12/11/43.

Temporal lobectomy Impairment index: 0.8

40. A 55-year-old American-born male; occupation: auto mechanic. Patient entered UCC 10/26/42 with a history of convulsive attacks followed by paralysis for seven months. Operation performed 10/29/42 by Dr. Walker involved osteoplastic craniotomy, partial removal of glioma and left parietal lobe. Recovery uneventful; patient discharged from hospital 11/8/42. Preoperative I.Q., 114 (Stanford-Binet, Form L).

Parietal lobectomy Impairment index: 0.6

41. A 40-year-old American-born female; occupation: housewife. Entered UCC 4/28/45 with a history of nervousness and dulling of memory for four to five weeks; two dizzy spells in five weeks; unsteady gait for four weeks; headaches for three weeks; diplopia for three weeks. Operation performed 5/10/45 by Dr. Jack Woolf involved bilateral temporal-parietal trepanation, partial left craniectomy. Recovery uneventful; patient discharged from hospital 5/20/45.

Intracerebral hematoma, parieto-occipital region Preoperative index: 0.7

42. A 50-year-old American-born male of German descent; occupation: farmer. Entered UCC 2/8/44 with a history of convulsions for seven years, starting in the right arm, and with weakness of right arm and leg, progressive, seven years. Operation performed 2/29/44 by Dr. Walker involved secondary osteoplastic craniotomy, removal of meningioma from right frontal lobe. Recovery uneventful; patient discharged from hospital 3/14/44.

Frontal lobectomy Impairment index: 0.8

43. A 52-year-old American-born male of Danish descent; occupation: service station attendant. Entered UCC 7/18/44 with a history of headaches for several years and dizziness and weakness for three months. Operation performed 8/1/44 by Dr. Walker involved first-stage osteoplastic craniotomy with biopsy of tumor tissue. Operation performed 8/10/44 by Dr. Walker involved secondary craniectomy, extirpation of meningioma from right frontal lobe. Operation performed 9/5/44 by Dr. Walker involved cranioplasty with tantalum plate. Recovery uneventful; patient discharged from hospital 10/9/44.

Frontal lobectomy Impairment index: 1.0

44. A 35-year-old American-born male; occupation: clergyman. Entered UCC 8/13/44 with a history of headaches for six weeks, dizziness for ten days, and poor memory for ten days. Operation performed 8/22/44 by Dr. Walker involved osteoplastic craniotomy, right frontal lobectomy, extirpation of glioblastoma. Patient expired in hospital 8/31/44.

Frontal lobectomy Preoperative impairment index: 0. 8

45. A 44-year-old American-born male; occupation: telephone engineer. Entered UCC 6/7/42 with a history of convulsions on 5/21/42 and on 6/7/42. Operation performed 6/18/42 by Dr. Walker involved osteoplastic craniotomy. Recovery uneventful; patient discharged from hospital 6/27/42.

Re-entered UCC 10/4/43 with a history of convulsions since May, 1942, about one per month. Operation performed 10/9/43 by Dr. Walker involved osteoplastic craniotomy, right frontal lobectomy, extirpation of glioma. Recovery uneventful; patient discharged from hospital 10/25/43.

Frontal lobectomy Impairment index: 1.0

46. A 39-year-old American-born male; occupation: liquor clerk. Entered UCC 2/10/43 with a history of headaches for eight months. Operation performed 2/12/43 involved osteoplastic craniotomy and left frontal lobectomy. Patient was severely disturbed for four days following operation, then recovery was uneventful; patient discharged from hospital 3/6/43.

Frontal lobectomy Impairment index: 1.0

47. A 16-year-old American-born male; occupation: student. Entered UCC 2/10/43

with a history of headaches for three months. Operation performed 2/25/43 by Dr. Walker involved evacuation of subdural hematoma over left frontal lobe. Recovery uneventful; patient discharged from hospital 3/5/43.

Hematoma in frontal lobe Impairment index: preoperative, 0.8; postoperative, 0.2

48. A 46-year-old American-born male of Russian descent; occupation: roll operator. Entered UCC 7/27/42 with a history of lack of control over right arm and leg for one month and pressure, headache, and dizziness on right side of head. Operation performed 8/8/42 by Dr. Walker involved osteoplastic craniotomy, cystoventriculostomy, evacuation of cystic glioma in left frontal lobe. Recovery uneventful; patient discharged from hospital 8/16/42.

Re-entered UCC 12/5/43 with a history of dull generalized headache for one year. Operation performed 12/9/43 by Dr. Walker involved secondary osteoplastic craniotomy, partial removal of brain tumor. Recovery uneventful; patient discharged from hospital 12/18/43.

Frontal lobectomy Impairment index: 0.8

49. A 21-year-old American-born male; occupation: unemployed. Entered UCC 8/18/43 with a history of major and minor convulsions two times per week since 1941. Operation performed 8/26/43 by Dr. Walker involved removal of cerebral scar from left frontal lobe, cranioplasty. Recovery uneventful; patient discharged from hospital 10/11/43. Preoperative I.Q., 99 (Stanford-Binet, Form L).

Frontal lobectomy Impairment index: 0.5

50. This patient came to our attention through the courtesy of Dr. Spafford Ackerly of Louisville, who had previously published a psychiatric study of her (2). A 48-year-old Hungarian-born female of Hungarian descent; occupation: housewife. Entered UCC 8/9/44 with a history of operations by Dr. Spurling of Louisville for removal of meningioma of the olfactory groove in 1933 after symptoms of headaches for three years, vomiting for six weeks, and loss of vision in the left eye. Has had no headache or vomiting since 1933. Personality since operation: shows lack of tact in dealings with friends, laughs and cries easily, predominantly euphoric, untidy, described as having a one-track mind, very religious. Memory excellent. No operation. Patient discharged from hospital 8/15/44.

Bilateral frontal lesion Impairment index: 1.0

APPENDIX B

DIAGRAMS OF BRAIN LESIONS OF CEREBRAL LOBECTOMIES

A diagram of the cerebral lesion in each of the cases just described is presented in the following figures. Each lesion is portrayed as visualized at the terminal stages of the neurosurgical operation. For a description of the method employed in preparing the diagrams and of the brain-lesion charts on which they are projected see chapter xiv and the writer's original paper (201). The diagrams are self-explanatory as to the location of the cerebral lesions in three dimensions. Extirpated, surgically removed areas are indicated in the original maps, which are one-third of life-size, by crosshatching; pathological tissue visualized during the operation but not removed is indicated by stippling. In the further reduction of these diagrams presented here, these two conditions are differentiated by shades of gray.

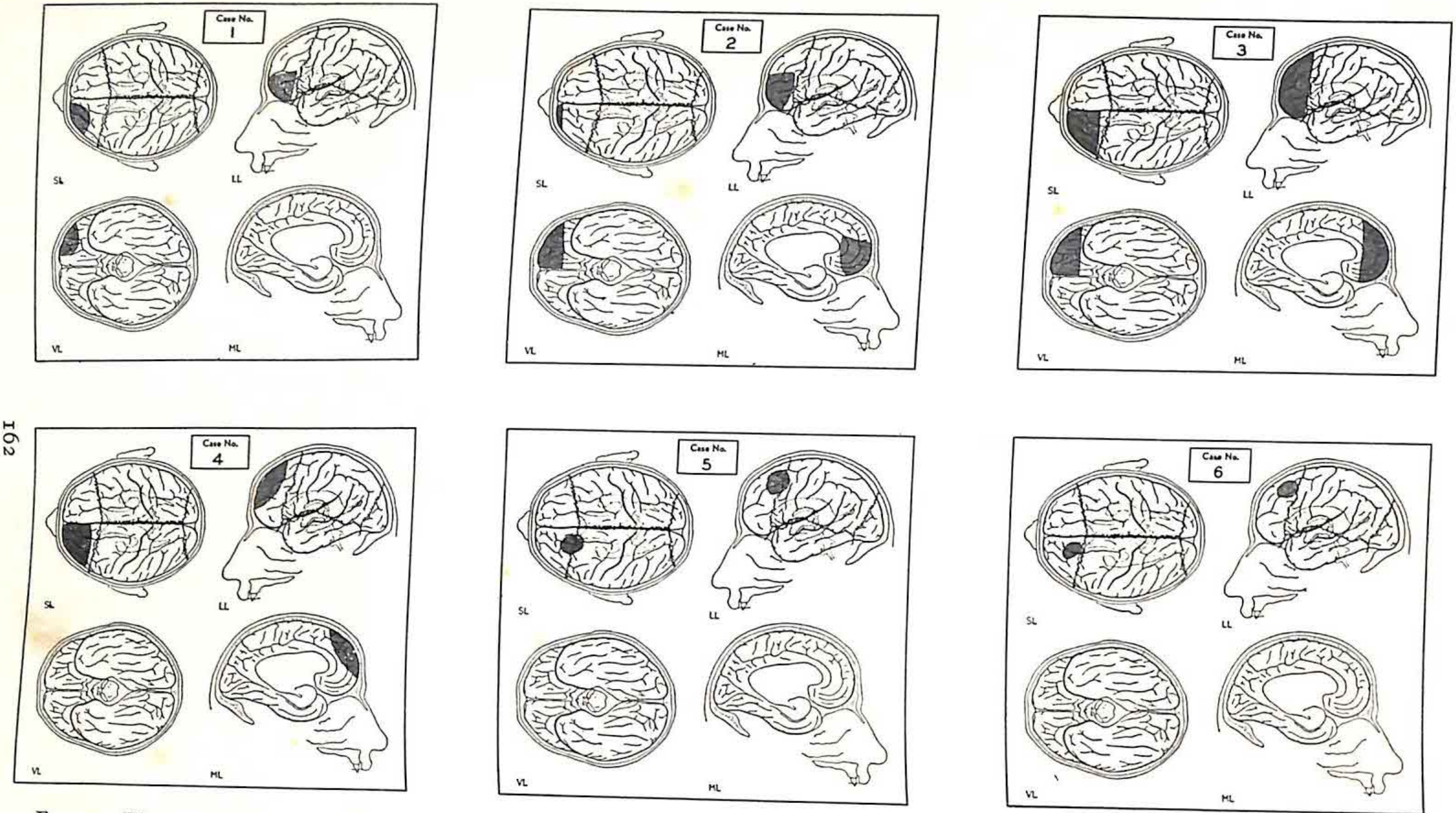


FIG. 20.—Diagrammatic representation to scale of the extent of the cerebral lesion in cases 1–6. For a percentage value for the lesion in the various cases shown in this and in the following figures, see Table 16, Appen. D. The general localization of the primary lesion is given directly by the four standard views of each diagram which are, respectively, superior, lateral, ventral, and medial.

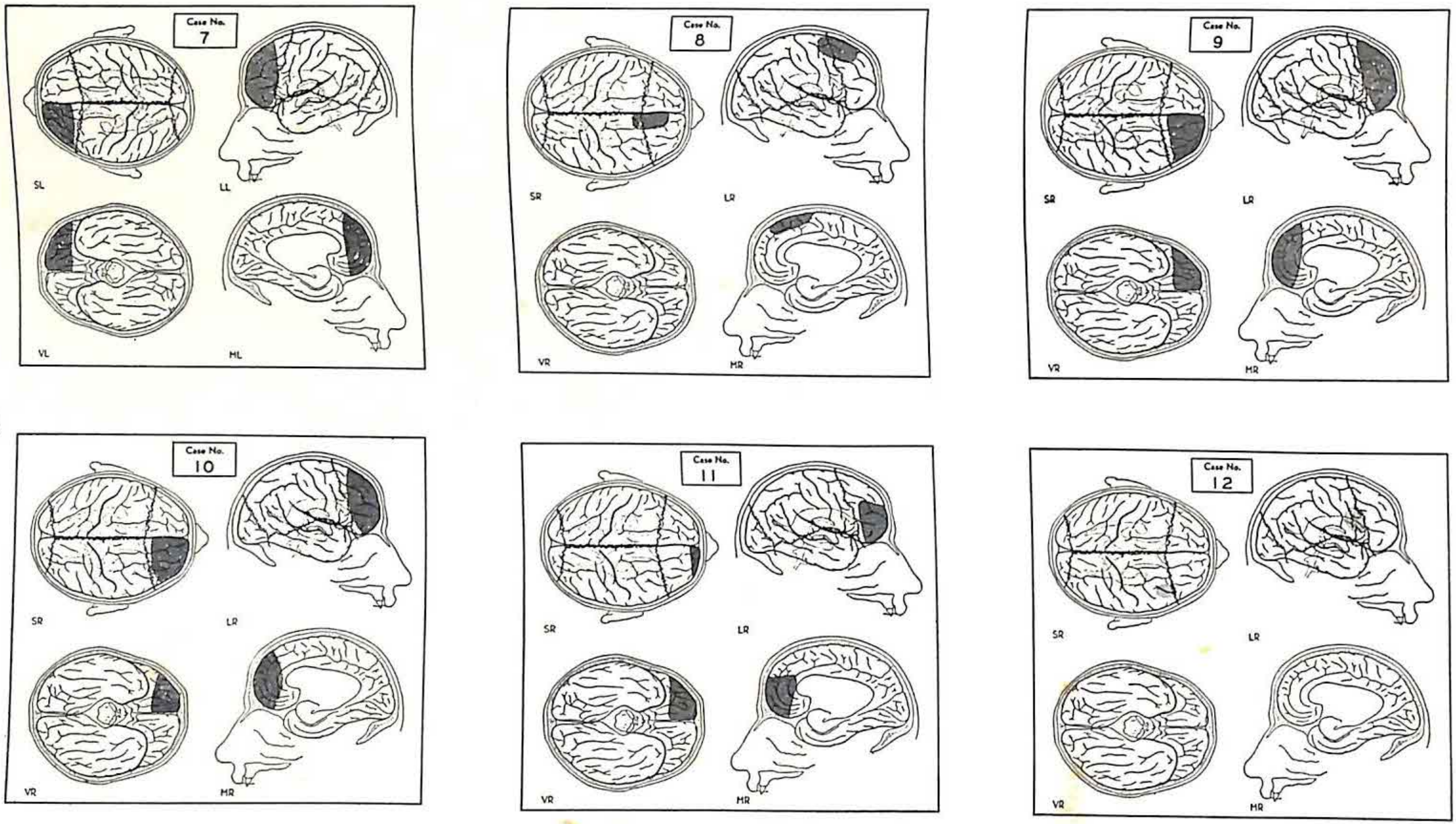


FIG. 21.—Diagrammatic representation to scale of the extent of the cerebral lesion in cases 7–12

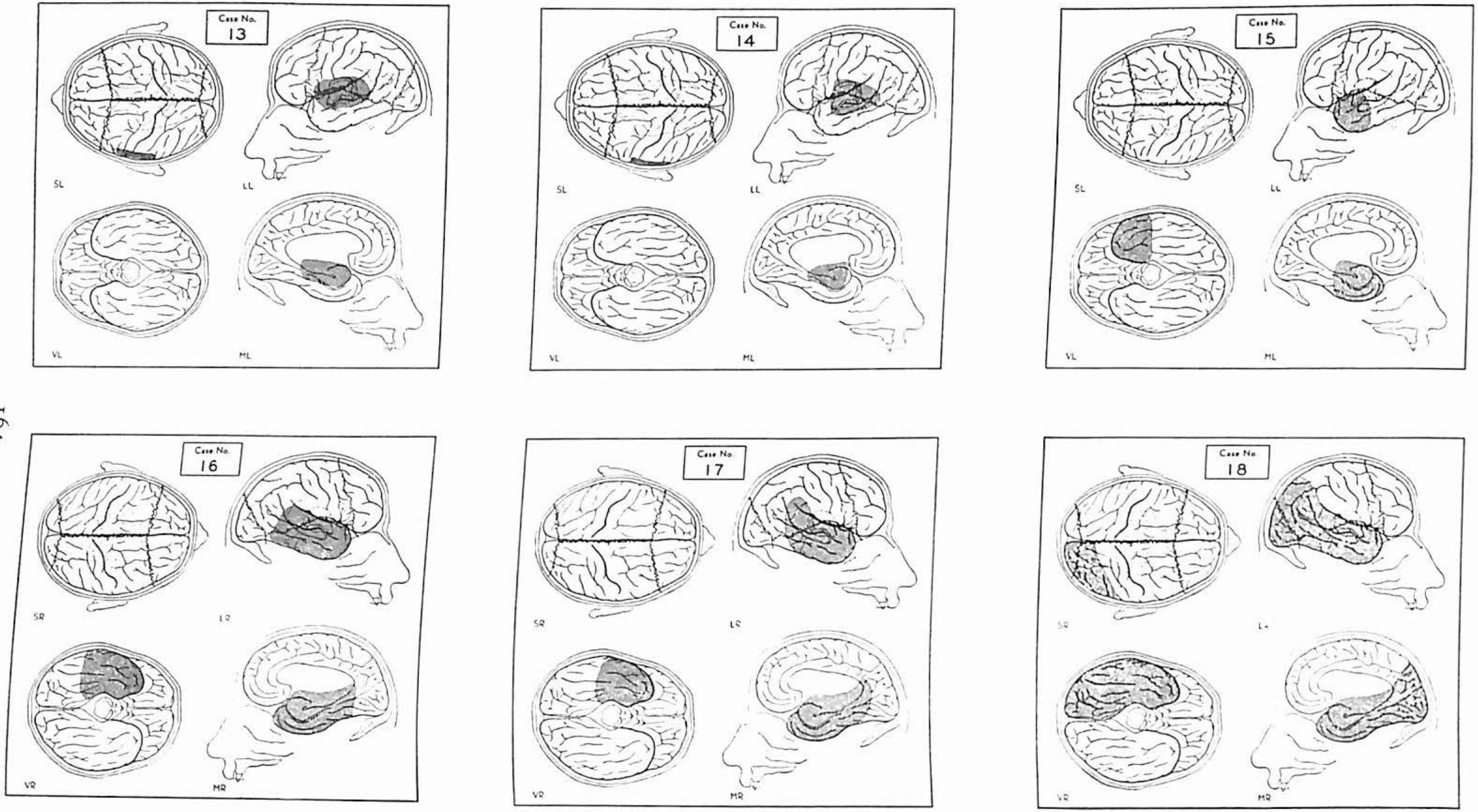


FIG. 22.—Diagrammatic representation to scale of the extent of the cerebral lesion in cases 13–18

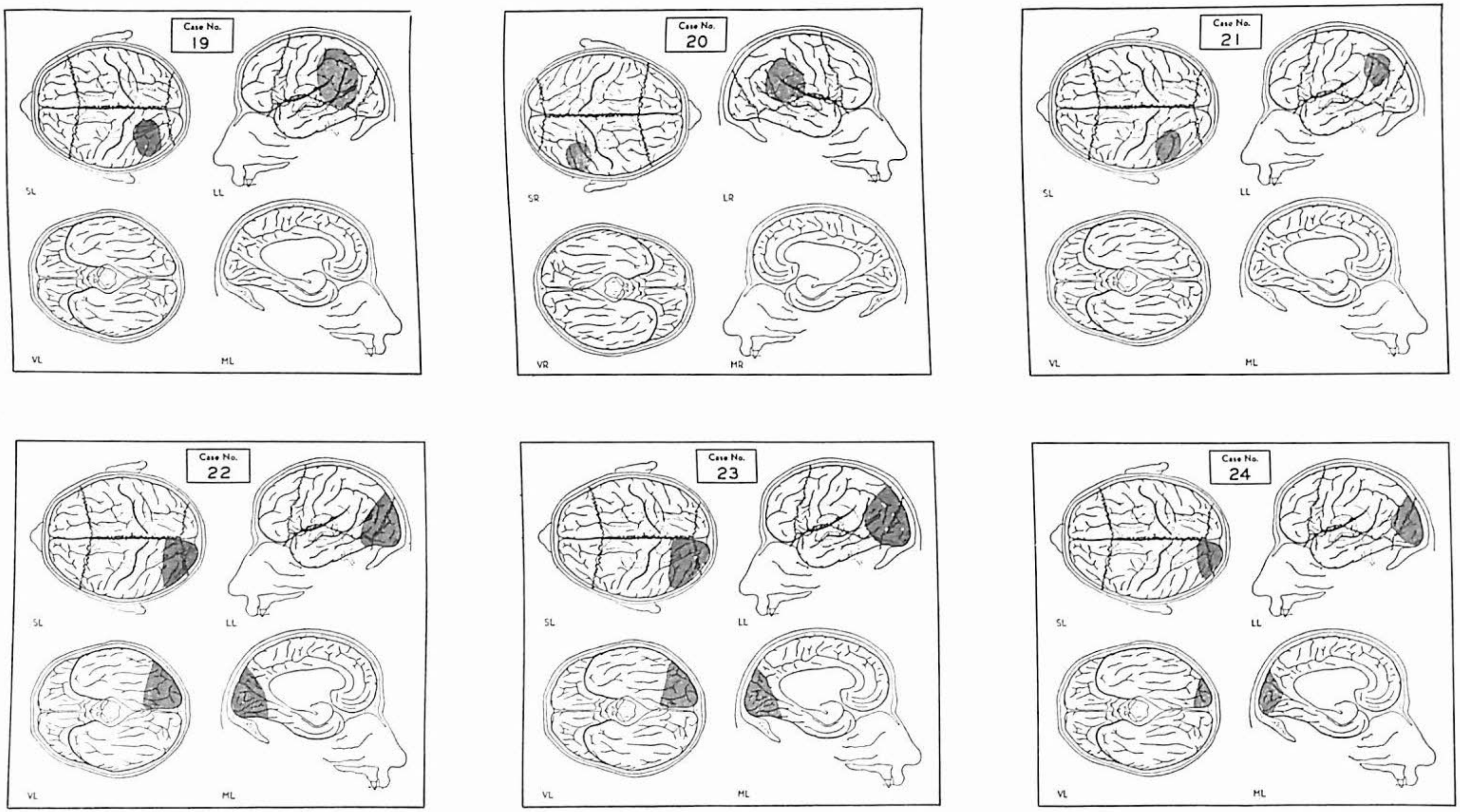


FIG. 23.—Diagrammatic representation to scale of the extent of the cerebral lesion in cases 19–24

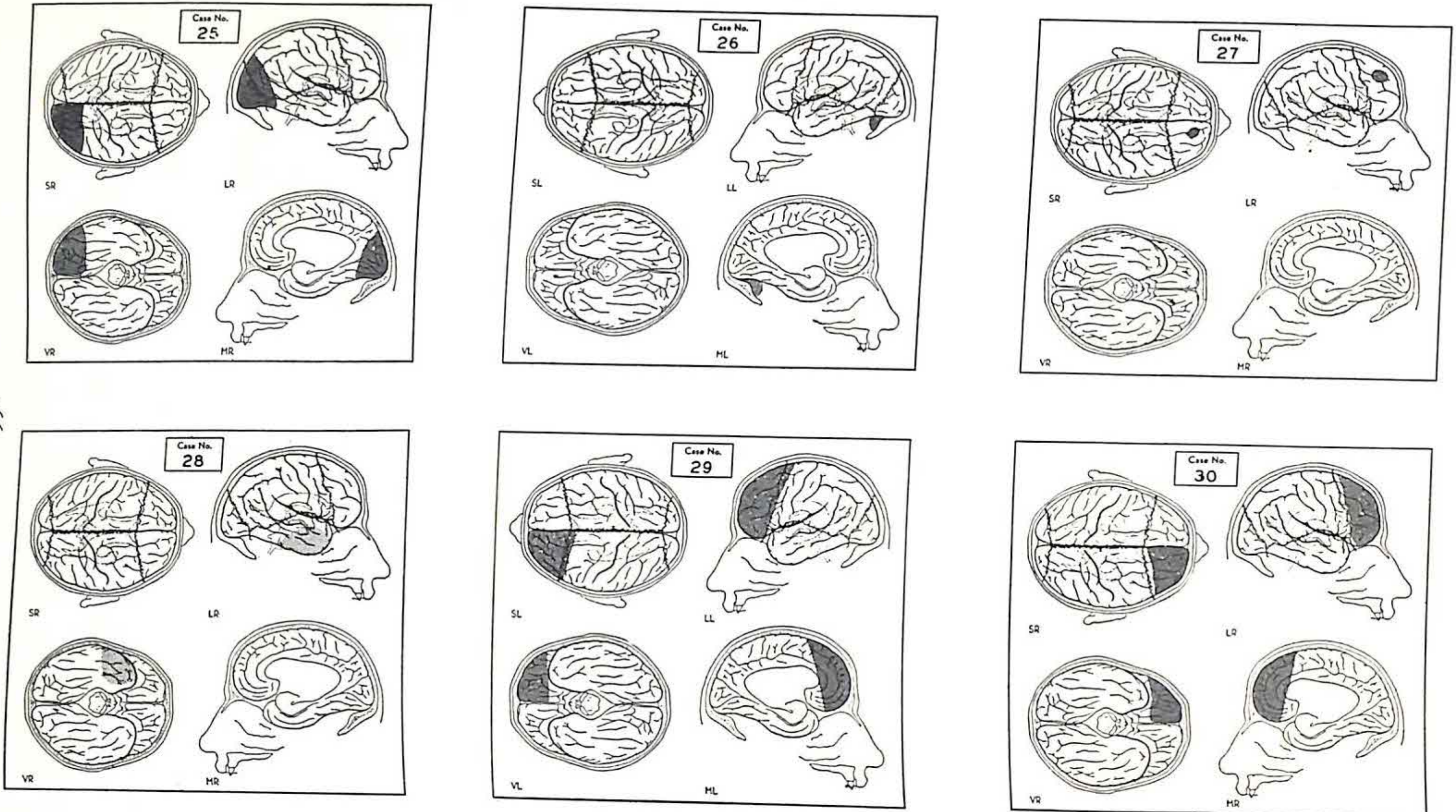


FIG. 24.—Diagrammatic representation to scale of the extent of the cerebral lesion in cases 25–30

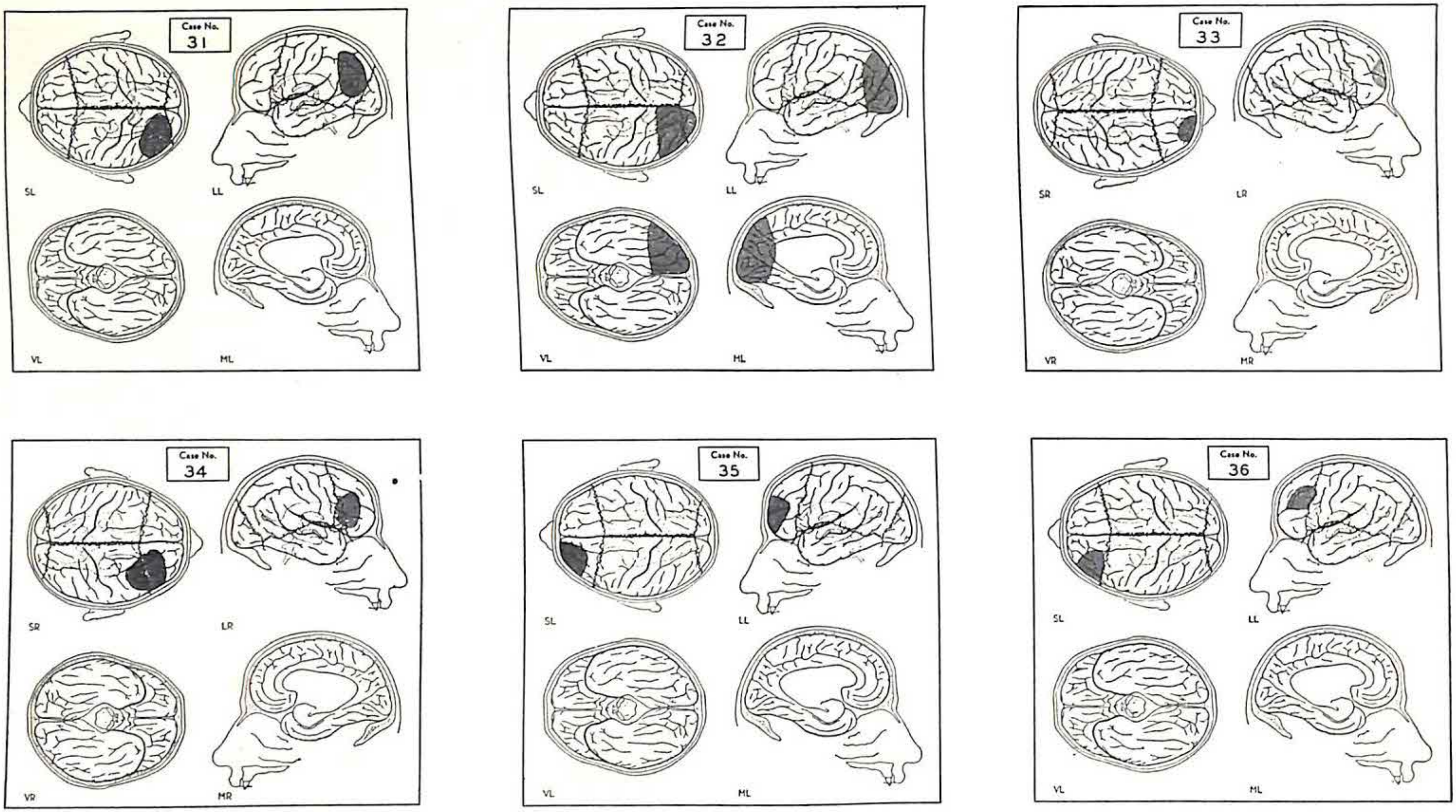


FIG. 25.—Diagrammatic representation to scale of the extent of the cerebral lesion in cases 31–36

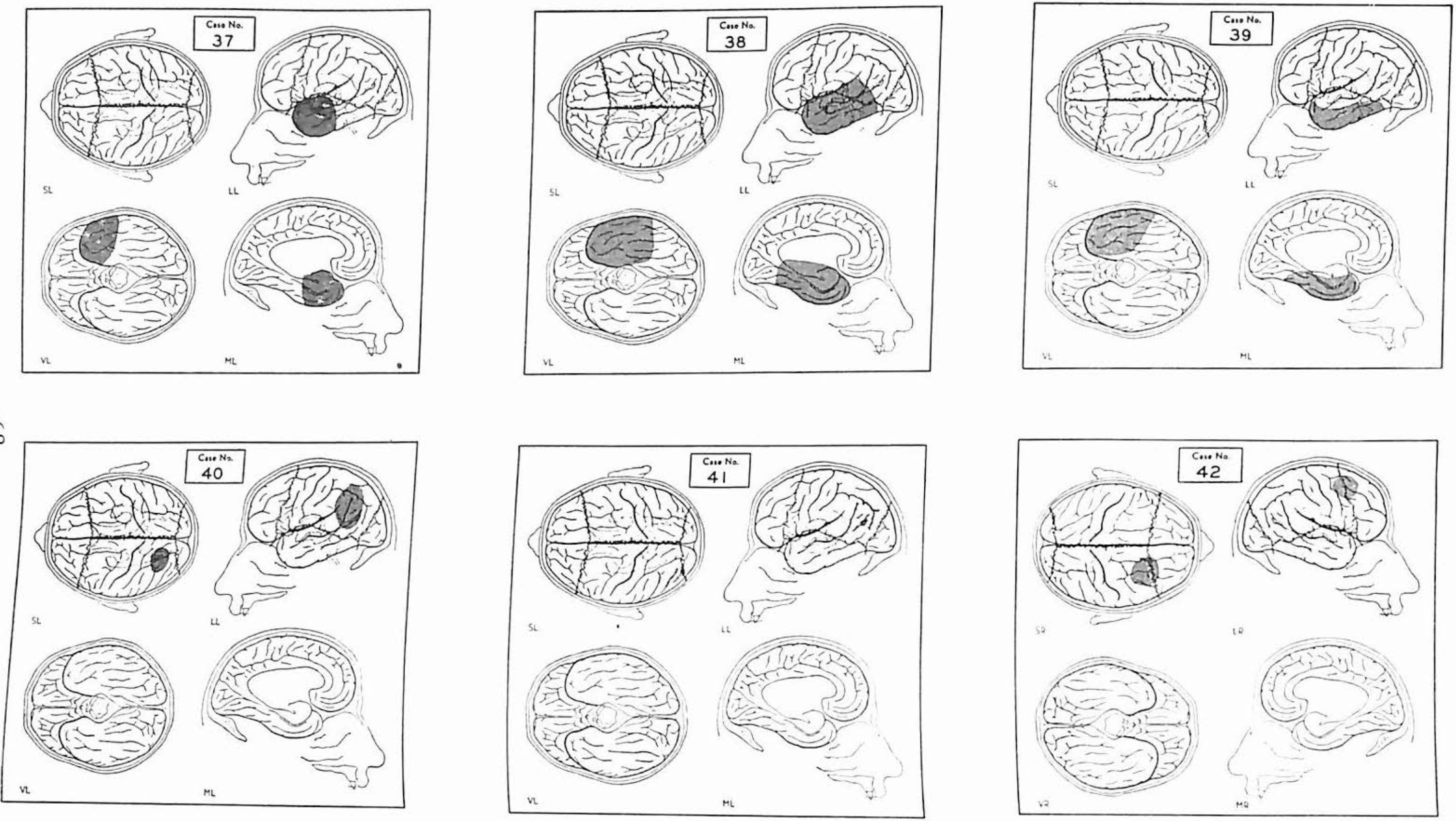


FIG. 26.—Diagrammatic representation to scale of the extent of the cerebral lesion in cases 37–42

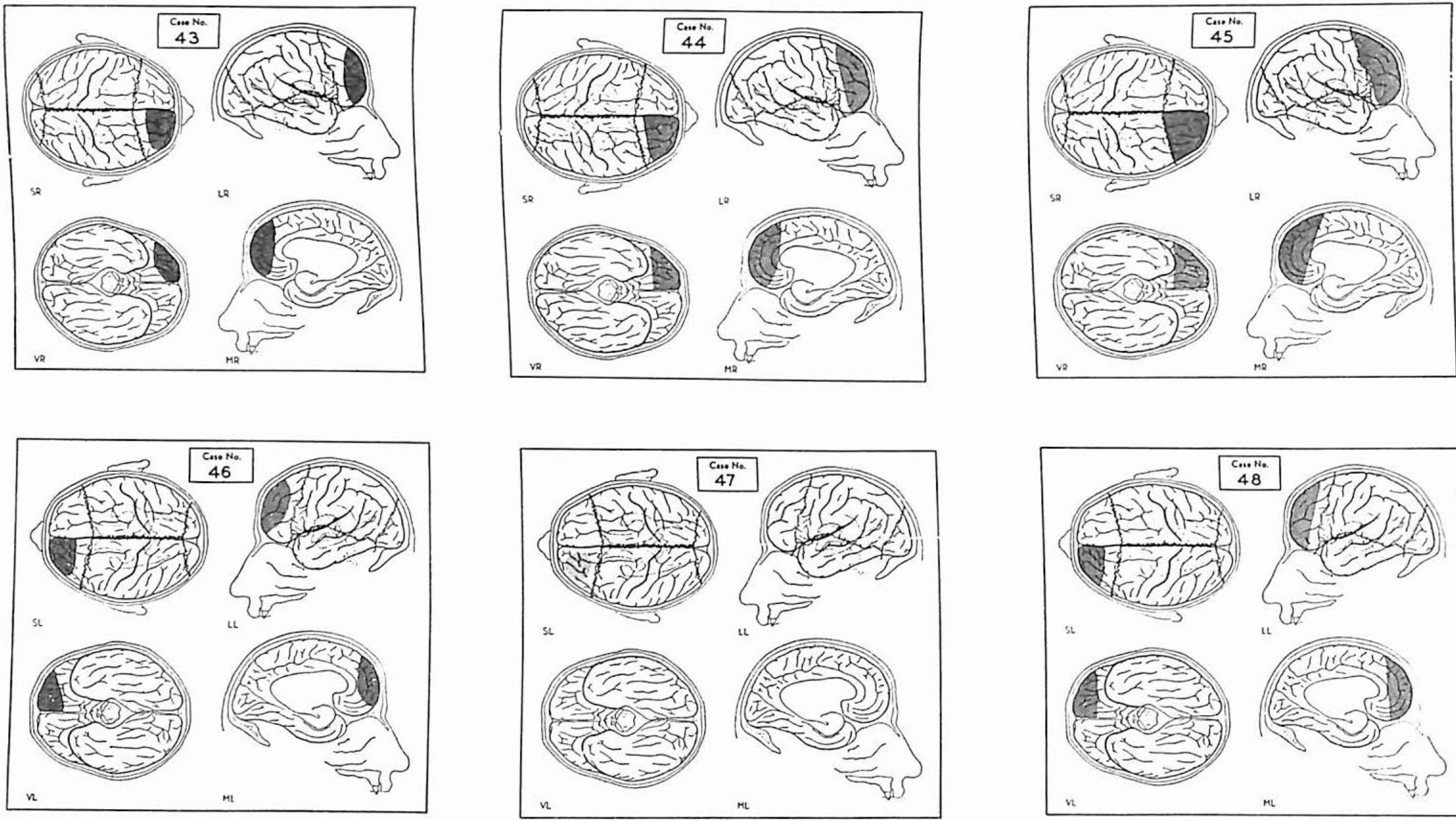
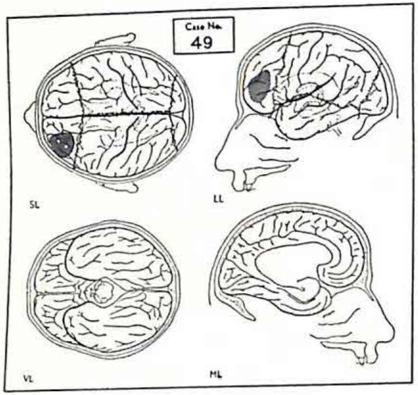
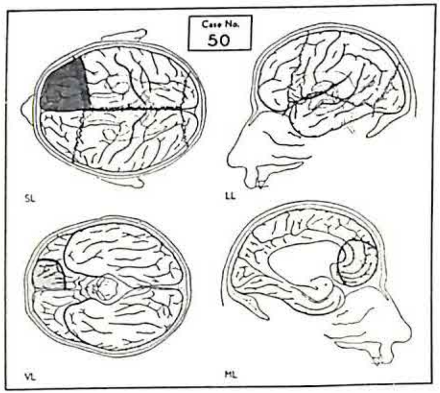


FIG. 27.—Diagrammatic representation to scale of the extent of the cerebral lesion in cases 43–50





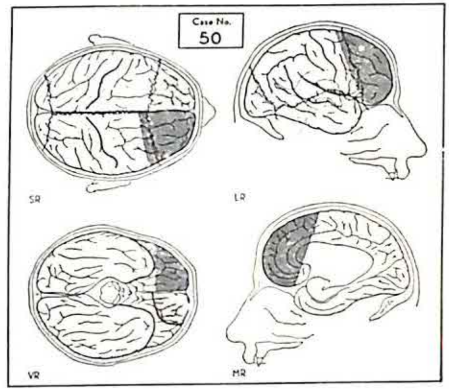


FIG. 27.—*Continued*

APPENDIX C

QUANTITATIVE INDICATORS

Some of the quantitative indicators employed in this study are described here for the first time. Some represent adaptations of test materials originally developed by other investigators. The rest are standardized tests selected to round out our battery. It is hoped that in such instances proper acknowledgments have been made. Where an indicator has been developed in this laboratory, the writer's name has been attached. This seems to offer one way in which the writer can assume responsibility for prescribed sets of test conditions.

A detailed technical description of each of the twenty-seven indicators in our battery seemed unsuited for this volume. It will, however, serve our present purposes to list these indicators by number and by name and to give a brief description of each.

1. *Carl Hollow-Square Test.*—This is a standardized performance test for general intelligence, which yields an I.Q. It was chosen for this study as a nonverbal test which had been standardized on hospital populations.

2. *Halstead Category Test.*—This test involves the ability of the subject to "abstract" various organizing principles, such as "size," "shape," "color," etc., from a series of 336 stimulus figures presented visually and serially by means of a multiple-choice projection apparatus.

3. *Halstead Flicker-Fusion Test.*—The basic unit of this test is modified from a Type 631-B General Radio Strobotac as an electronic source of variably intermittent light. Low-level critical-fusion frequency of the subject is measured in ten trials, the last five of which are regarded as critical.

3*a*. *Average Deviation of Critical-Fusion Frequency.*—This variable consists of the average deviation of the five critical trials in the preceding test.

4. *Halstead Performance Test (Speed).*—This is the writer's modification of the Seguin-Goddard form board test. The board is presented at an angle of 70° with reference to the horizontal. At no time is the subject permitted to see the form board or blocks. Three timed trials are given: the first with the dominant hand only, the second with the other hand, and a third with both hands.

5. *Halstead Performance Test (Recall).*—Following three timed trials without vision, the subject is asked to draw an outline of the board as he imagines it to be, including the shapes of the various blocks and their location on the board. The number of shapes out of ten accurately recalled constitutes the score on this test.

6. *Halstead Performance Test (Localization).*—The number of shapes out of ten accurately localized in the drawing made in Test 5 constitutes the score on this test.

7. *Multiple-Choice Ink Blots.*—In this test, the ten standard Rorschach ink-blot figures are presented from lantern slides (2×2 inches). After preliminary explorations the scoring technique proposed by Harrower-Erickson (213) was finally adopted.

8. *Minnesota Multiphasic Personality Inventory.*—This test consists of 550 statements, bearing on physical and mental health, which are printed on separate cards. The subject sorts these cards into "True," "False," and "Cannot Say" categories as he thinks they apply to him. A profile score is obtained.

9. *Henmon-Nelson Tests of Mental Ability.*—The one employed is a paper-and-pencil test of verbal intelligence, which yields an I.Q. It is not widely used as an individual test, an advantage for our purposes. None of our subjects had ever taken the test previously.

10. *Hunt-Minnesota Test for Organic Brain Damage.*—This somewhat ambitiously entitled test consists of a vocabulary test, an associative memory test for visual designs, and an associative memory test (auditory) for word pairs. It was included for purposes of comparison with other indicators.

11. *Halstead Schematic Face Test.*—This test consists of nine schematic faces from the series developed by Brunswik and Reiter (66), presented in two groupings on a card 8×10 inches. For each grouping, the subject is asked to select freely the "gay face," the "sad face," the "young face," etc., for a total of fourteen judgments. The score is the total number of choices which fall outside popular or conventional categories.

12. *Seashore Measures of Musical Talent.*—These measures constitute a convenient source of standardized auditory stimuli from phonograph records. Separate discrimination subtests are provided for pitch, loudness, time, timbre, rhythm, and tonal memory.

13. *Speech-Sounds Perception Test.*—This is a modification of a test, on phonograph records, supplied to the writer by Professor Louis D. Goodfellow. It consists of sixty spoken speech sounds which are nonsense-syllable variants of the *ee* digraph, presented in multiple-choice form.

14. *Halstead Finger-Oscillation Test.*—This test determines the maximum rate at which the subject can oscillate the extended index finger of the dominant hand by requiring him to activate the lever arm of a mechanical counter. Five trials are given and averaged.

15. *Halstead Time-Sense Test* (*Vision*).—This test involves the estimation of a ten-second interval by starting and stopping an electric clock. Memory tests are interspersed with visual trials in the sequence V V M V M V. Forty visual trials are given in groups of ten. The score is the average deviation from ten seconds.

16. *Halstead Time-Sense Test* (*Memory*).—This variable comprises twenty trials on Test 15, performed without the aid of vision. The average deviation from ten seconds is the basis of scoring.

17. *Halstead Dynamic Visual Field Test* (*Central Form*).—In this test the subject is required to make a form and color discrimination at the fovea at the same instant that he must detect the presence of a graduated, circular patch of neutral light somewhere in the peripheral visual field. The targets are presented automatically for twenty milliseconds. The subject records on prepared diagrams what he has seen after each exposure. The score for "central form" is the number of errors in discrimination out of 120 items.

18. *Halstead Dynamic Visual Field Test* (*Central Color*).—This is the color component of the central-form targets exposed in Test 17. The score is the number of errors in discrimination.

19. *Halstead Dynamic Visual Field Test* (*Peripheral Component*).—This is the peripheral target component for the items exposed in Test 17. Ten per cent of the items are control exposures—there is no associated peripheral target. The score on this component is the number of errors (targets not seen) out of 108 possibilities.

20. *Manual Steadiness Test.*—In this test the subject is required to insert a metal stylus into holes of progressively smaller diameter, at the same time avoiding contact with the metal boundary of each hole. The score is the total number of contacts at five seconds per hole for nine holes.

21. *Halstead-Brill Audiometer.*—This is a carefully calibrated device for measuring hearing loss in decibels through the frequency range from 64 to 22,000 cycles per second. A profile audiogram is mapped during the test.

22. *Halstead Aphasia Test.*—This is a screening test for aphasia, agnosia, and apraxia. The test materials are printed on a cardboard wheel, and responses are recorded on a specially prepared chart.

23. *Shlaer-Hecht Anomaloscope.*—This is a color-vision test for red-green blindness, made available to this laboratory prior to its publication through the courtesy of Dr. Selig Hecht.

24. *Halstead Weight-Discrimination Test.*—This test is designed to measure the strength of the size-weight illusion.

25. *Halstead Color Gestalt Test.*—This is a grouping test, using the Ishihara color plates mounted individually under glass and the instruction: "Place those together that seem to belong together." This procedure is repeated until the new-grouping principles available to the subject are exhausted. No satisfactory basis of scoring this test has yet been developed.

26. *Halstead Closure Test.*—This test consists of a photograph of an outdoor scene in which a cow is well camouflaged. The task is to find the cow. Time up to five minutes is scored for success.

Detailed medical histories for each patient were available to the writer. These included visual acuity (Snellen) determinations and the data from complete neurological examinations.

APPENDIX D

ORIGINAL DATA FOR CONTROL SUBJECTS AND FOR CEREBRAL LOBECTOMIES

TABLE 14*

CRITERION SCORES†

	Indicators									
	2	3	3*a*	4	5	6	12	13	14	16
Criterion score......	>80	<21.0	<.7	>15.6	<6	<5	>35	>20	<51	>260

* In computing the impairment index, given in the following tables, the criterion scores shown have been employed. The symbol > is used to mean "greater than" and < to mean "less than."

† For a description of the nature of each score see Appen. C.

TABLE 15*

Subjects Used in Control Studies

Subject	Age	Indicators										Index
		2	3	3a	4	5	6	12	13	14	16	
		Civilian Controls										
I. R. M.	17	24	24.8	2.2	6.0	10	10	11	8	53	248.3	0.0
II. M. M.	25	32	28.2	1.1	5.2	9	9	31	7	61	226.6	.0
III. D. J.	37	39	27.2	1.1	10.5	8	7	20	3	57	339.0	.1
IV. M. C.	18	16	20.4	1.2	7.6	9	3	28	4	53	316.1	.3
V. E. M. C.	43	21	30.5	1.8	5.6	9	4	14	3	59	239.5	.1
VI. H. Z.	17	23	17.9	.7	12.4	9	8	12	14	55	304.6	.2
VII. G. D.	20	12	22.7	1.8	8.8	10	10	25	6	49	242.2	.1
VIII. O. J.	21	28	23.0	1.0	8.5	6	2	14	12	61	135.9	.1
IX. R. J.	32	10	24.4	1.5	9.8	9	7	11	11	54	155.0	.0
X. J. W.	23	.....	21.5	1.3	5.9	9	7	14	5	47	151.1	.1
XI. E. W.	24	37	21.8	1.0	11.1	6	3	15	5	57	455.9	.2
XII. R. B.	14	46	20.1	.9	10.5	8	3	9	15	48	133.5	.3
XIII. L. B.	50	24	19.6	.6	19.6	8	8	16	10	54	114.9	.3
XIV. F. B.	22	36	22.5	.3	11.7	8	5	22	9	52	180.5	0.1
Average	25.9	26.8	23.2	1.2	9.5	8.4	6.1	17.3	8.0	54.3	231.6	0.14
		Military Controls										
XV. E. M.	22	35	28.3	1.1	15.8	7	7	17	18	51	190.9	0.1
XVI. A. K.	32	55	21.5	1.3	6.3	9	7	16	8	60	216.0	.0
XVII. M. L.	38	75	23.8	1.3	11.0	6	1	6	10	57	154.1	.1
XVIII. E. M.	30	55	25.9	.8	13.8	9	8	34	.....	53	89.8	.0
XIX. C. E.	25	45	27.2	.8	13.2	8	7	45	.....	55	219.3	.1
XX. L. H.	31	59	22.2	1.2	11.3	8	5	21	8	56	164.4	.0
XXI. M. T.	24	29	26.0	.9	8.6	8	7	18	17	57	134.3	.0
XXII. K. D.	31	60	23.8	.9	11.3	7	3	43	20	55	120.3	.2
XXIII. F. G.	36	51	24.2	1.3	15.9	9	6	40	19	51	123.7	.2
XXIV. W. P.	23	44	25.0	1.8	13.1	7	5	19	16	63	118.0	0.0
Average	29.2	50.8	24.8	1.1	12.0	7.8	5.6	25.9	14.5	55.8	153.1	0.07
		Miscellaneous Controls										
XXV. C. K.	27	35	13.3	.6	12.7	8	1	31	12	59	240.0	0.3
XXVI. R. C.	33	93	17.7	1.5	13.3	7	5	15	4	54	547.5	.3
XXVII. I. E. 1	39	18	18.9	.3	13.3	7	2	16	3	59	236.8	.3
XXVIII. I. E. 2	39	20	17.3	.2	12.0	10	7	48	8	55	122.8	.3
XXIX. F. W. 1	28	16	22.2	.9	12.8	8	7	24	6	56	694.3	.1
XXX. F. W. 2	28	27	22.4	1.4	5.9	9	7	19	9	56	128.6	0.0
Average	32.3	34.8	18.6	.8	11.7	8.2	4.9	25.5	7.0	56.5	328.3	0.22

* Certain minor errors in calculating the impairment index for each subject shown in Tables 15 and 16 which appeared in the first printing of this monograph are corrected in this second printing. It was found that the averages were unchanged by these corrections.

TABLE 16

NEUROSURGICAL PATIENTS

SUBJECT	AGE	INDICATORS 2	3	3a	4	5	6	12	13	14	16	INDEX	TYPE	TOTAL PER CENT LESION
							Frontal Lobectomies							
1...	48	......	17.2	.3	28.0	3	1	37	.....	44	641.5	1.0	L.F.	3.38
2...	55	......	18.6	.4	19.2	5	3	.....	.....	41	310.2	1.0	L.F.	5.14
3...	58	......	19.2	.3	.....	.....	.....	.....	.....	.....	......	....	L.F.	10.69
4...	32	......	17.6	.2	21.3	6	4	21	.....	39	417.3	0.8	L.F.	5.41
5...	36	......	20.1	.3	17.0	6	3	18	.....	45	......	0.7	L.F.	2.30
6...	51	105	15.8	.4	58.0	4	0	51	28	49	380.9	1.0	L.F.	1.35
7...	53	......	14.4	.5	28.3	3	1	39	.....	41	581.2	1.0	L.F.	10.69
8...	32	......	16.7	.5	33.2	4	4	19	.....	43	354.3	0.9	R.F.	3.65
9...	47	......	22.6	1.1	19.4	3	2	42	.....	48	161.5	0.6	R.F.	12.17
10...	31	85	21.2	1.3	.....	.....	.....	.....	.....	46	976.6	0.6	R.F.	9.74
11...	46	......	18.2	.3	15.0	5	4	38	.....	50	451.3	0.9	R.F.	6.49
12...	33	80	17.5	1.1	11.6	6	4	51	23	61	296.2	0.5	R.F.	1.35
27...	14	......	17.4	.9	20.6	8	7	.....	.....	47	341.7	0.6	R.F.	0.67
29...	46	......	14.8	.2	23.5	4	2	43	.....	41	255.4	0.9	L.F.	14.61
30...	63	......	14.2	.8	.....	.....	.....	.....	.....	37	18.8	0.5	R.F.	12.31
33...	16	87	15.1	.3	.....	.....	.....	47	.....	45	774.4	1.0	R.F.	1.35
34...	44	......	17.3	.4	19.4	5	3	41	.....	53	206.1	0.8	R.F.	4.05
35...	28	117	13.8	.3	13.3	7	7	48	14	45	320.9	0.6	L.F.	2.70
36...	49	......	20.1	.6	23.0	5	2	19	.....	46	......	0.9	L.F.	2.30
42...	50	79	16.7	.5	12.0	5	1	.....	.....	35	431.6	0.8	R.F.	2.57
43...	52	102	18.3	.3	20.2	3	0	57	36	48	500.8	1.0	R.F.	7.71
44...	35	......	.....	....	18.3	6	1	.....	.....	36	288.6	0.8	R.F.	10.14
45...	44	......	.....	....	.....	.....	.....	.....	23	32	4,540.1	1.0	R.F.	12.17
46...	39	......	.....	....	23.1	4	0	.....	.....	31	1,048.1	1.0	L.F.	6.63
47...	16	105	16.7	.4	23.5	5	3	31	8	46	290.6	0.8	L.F.	Hematoma
48...	46	40	17.2	.4	22.8	6	0	.....	.....	48	886.4	0.8	L.F.	8.11
49...	21	109	17.2	.3	8.5	6	3	34	11	40	157.1	0.5	L.F.	2.43
50...	48	131	11.7	.6	20.2	4	3	44	.....	47	......	1.0	Bi.F.	28.27
Av.	40.4	94.5	17.2	.5	21.7	4.9	2.5	37.8	20.4	43.9	609.7	.8	......	6.98
							Nonfrontal Lobectomies							
18...	31	......	12.6	.2	.....	.....	.....	15	.....	51	143.0	0.4	R.P.O.	32.47
19...	19	16	28.3	1.6	10.2	9	8	14	6	43	146.2	0.1	L.P.	5.54
20...	28	......	26.2	1.5	13.3	8	7	38	.....	39	187.3	0.3	R.P.	4.46
21...	21	......	19.1	1.2	14.7	7	5	20	.....	54	176.0	0.1	L.P.	2.70
31...	35	......	23.0	1.2	.....	.....	.....	.....	.....	.....	......	....	L.P.O.	4.87
40...	55	......	.....	....	28.8	6	1	.....	.....	42	11.7	0.6	L.P.	2.70
41...	40	85	16.0	1.2	27.6	5	0	56	26	54	143.0	0.7	L.P.	Hematoma
13...	19	......	23.4	1.6	12.3	7	6	15	.....	49	357.0	0.3	L.T.	5.14
14...	35	......	26.0	1.3	19.7	8	7	21	.....	36	150.1	0.3	L.T.	4.73
15...	33	......	17.2	1.4	13.4	8	8	16	.....	46	172.0	0.3	L.T.	9.33
16...	51	......	12.6	1.8	11.7	7	5	17	.....	52	481.0	0.3	R.T.	17.32
17...	36	......	24.3	1.1	13.2	8	6	19	.....	57	391.2	0.1	R.T.	16.50
28...	46	......	27.6	.8	9.4	8	8	14	.....	53	203.4	0.0	R.T.	4.60
37...	39	37	24.2	1.2	12.6	7	4	26	10	43	304.7	0.3	L.T.	8.52
38...	47	179	10.9	.6	46.3	6	1	60	37	16	1,023.9	0.9	L.T.	16.77
39...	26	......	18.5	.4	12.0	7	4	45	21	48	356.8	0.8	L.T.	10.96
22...	25	93	24.3	1.0	13.1	7	5	43	13	69	240.9	0.2	L.O.	11.23
23...	35	......	27.5	1.4	12.6	7	6	19	.....	56	184.2	0.0	L.O.	12.04
24...	22	......	23.2	1.3	.....	.....	.....	.....	.....	.....	......	....	L.O.	5.27
25...	27	43	17.3	1.0	10.8	8	4	26	19	52	138.6	0.2	R.O.	10.01
32...	41	53	24.2	1.8	13.8	7	5	31	.....	49	167.2	0.1	L.O.	14.88
26...	15	......	28.4	1.6	11.3	9	9	17	.....	58	143.0	0.0	Cerebellar	......
Av.	33.0	72.3	21.7	1.2	16.1	7.3	5.2	26.0	18.0	[illegible]				

TABLE 17

AVERAGE IMPAIRMENT INDEX AND AVERAGE SCORE ON EACH COMPONENT SUBTEST FOR CONTROL GROUP (CIVILIAN AND MILITARY COMBINED) FRONTAL LOBECTOMIES, AND NONFRONTAL LOBECTOMIES*

Indicator	Controls Average X_1	Frontal Lobectomies Average X_2	Nonfrontal Lobectomies Average X_3	Significance of Difference, P		
				X_1, X_2	X_1, X_3	X_2, X_3
2	37.22	94.50	72.30	$t=8.03$ $P<.001$	$t=2.78$ $P<.006$	$t=1.20$ $P<.033$
3	23.44	17.20	21.70	$t=8.47$ $P<.001$	$t=1.68$ $P<.056$	$t=3.72$ $P<.001$
3*a*	1.16	.50	1.20	$t=6.39$ $P<.001$	$t=.312$ $P<.379$	$t=6.50$ $P<.001$
4	10.56	21.70	16.10	$t=5.21$ $P<.001$	$t=2.73$ $P<.006$	$t=1.92$ $P<.046$
5	8.17	4.90	7.30	$t=8.99$ $P<.001$	$t=2.67$ $P<.007$	$t=6.46$ $P<.001$
6	5.92	2.50	5.20	$t=5.15$ $P<.001$	$t=.91$ $P<.189$	$t=3.83$ $P<.001$
12	20.88	37.80	26.90	$t=4.82$ $P<.001$	$t=1.55$ $P<.070$	$t=2.46$ $P<.022$
13	10.36	20.40	18.90	$t=3.45$ $P<.001$	$t=2.85$ $P<.005$	$t=.27$ $P<.384$
14	54.92	43.90	48.40	$t=7.14$ $P<.001$	$t=2.74$ $P<.006$	$t=1.80$ $P<.054$
16	198.90	609.70	256.10	$t=2.29$ $P<.016$	$t=1.17$ $P<.152$	$t=1.70$ $P<.062$
Impairment index	0.1	0.8	0.3	$t=17.06$ $P<.001$	$t=3.20$ $P<.002$	$t=7.94$ $P<.001$

* Where probability (P) is less than 5 per cent ($P<.05$), a significant difference is indicated (R. A. Fisher [133]).

REFERENCES AND BIBLIOGRAPHY

REFERENCES AND BIBLIOGRAPHY

1. ACH, N. Ueber die Willenstätigkeit und das Denken. Göttingen: Vandenhoeck & Ruprecht, 1905. Pp. x+294.
2. ACKERLY, S. Instinctive emotional and mental changes following prefrontal lobe extirpation, Am. J. Psychiat., **92**:712–92, 1935.
3. ADDISON, W. H. F., and DONALDSON, H. H. On the area of the sunken cerebral cortex as determined from the length and depth of selected sulci in three classes of human brains: scholars, hospital whites, hospital Negroes, J. Comp. Neurol., **57**:429–53, 1933.
4. ADRIAN, E. D. Brain mechanism, Science, **99**:353–57, 1944.
5. ADRIAN, E. D., and MATTHEWS, B. H. C. The Berger rhythm: potential changes from the occipital lobes in man, Brain, **57**:355–85, 1934.
6. ALEXANDER, F.; FRENCH, T. M.; *et al.* Psychoanalytic therapy. New York: Ronald Press, 1946. Pp. xiii+353.
7. ARMSTRONG, H. G. Principles and practices of aviation medicine. 2d ed. Baltimore: Williams & Wilkins, 1943. Pp. xiv+514.
8. BAILEY, P. Intracranial tumors. Springfield, Ill.: Charles C. Thomas, 1933. Pp. xxii+475.
9. BAILEY, P., and BREMER, F. A sensory cortical representation of the vagus nerve, J. Neurophysiol., **1**:405–12, 1938.
10. BAILEY, P., and SWEET, W. H. Effects on respiration, blood pressure, and gastric motility of stimulation of orbital surface of frontal lobe, J. Neurophysiol., **3**:276–81, 1940.
11. BARCROFT, J. The brain and its environment. New Haven: Yale University Press, 1938. Pp. vii+117.
12. BARD, P. A diencephalic mechanism for the expression of rage with special reference to the sympathetic nervous system, Am. J. Physiol., **84**:490–515, 1928.
13. ———. Central nervous mechanisms for emotional behavior patterns in animals, A. Research Nerv. & Ment. Dis., Proc., **19**:190–218, 1939.
14. BARTLEY, S. H. Temporal and spatial summation of extrinsic impulses with the intrinsic activity of the cortex, J. Cell. & Comp. Physiol., **8**:41–62, 1936.
15. ———. The basis of flicker in the visual field surrounding the test object, J. Exper. Psychol., **19**:342–50, 1936.
16. ———. The neural determination of critical flicker frequency, ibid., **21**:678–86, 1937.
17. ———. A central mechanism in brightness discrimination, Proc. Soc. Exper. Biol. & Med., **38**:535–36, 1938.
18. ———. Subjective flicker rate with relation to critical flicker frequency, J. Exper. Psychol., **22**:388–94, 1938.
19. ———. Subjective brightness in relation to the flash rate and the light-dark ratio, ibid., **23**:313–19, 1938.
20. ———. Some effects of intermittent photic stimulation, ibid., **25**:462–80, 1939.
21. ———. Some factors in brightness discrimination, Psychol. Rev., **46**:337–58, 1939.
22. ———. The relation between cortical response to visual stimulation and changes in the alpha rhythm, J. Exper. Psychol., **27**:624–39, 1940.

23. ———. Vision: a study of its basis. New York: D. Van Nostrand Co., 1941. Pp. xv+350.
24. ———. The features of the optic-nerve discharge underlying recurrent vision, J. Exper. Psychol., **30**:125–35, 1942.
25. ———. Conflict, frustration and fatigue, Psychosom. Med., **5**:160–63, 1943.
26. Bartley, S. H., and Bishop, G. H. The cortical response to stimulation of the optic nerve in the rabbit, Am. J. Physiol., **103**:159–72, 1933.
27. ———. A functional study of the nerve element of the optic pathway by means of the recorded action currents, Am. J. Ophth., **17**:995–1007, 1934.
28. ———. Optic nerve response to retinal stimulation in the rabbit, Proc. Soc. Exper. Biol. & Med., **44**:39–41, 1940.
29. ———. Some features of the optic-nerve discharge in the rabbit and cat, J. Cell. & Comp. Physiol., **19**:79–93, 1942.
30. Beck, S. J. Psychological processes in Rorschach findings, J. Abnorm. & Social Psychol., **31**:482–88, 1937.
31. ———. Rorschach's test I: basic processes. New York: Grune & Stratton, 1944. Pp. 223.
32. Bennett, A. E. An evaluation of the "shock" therapies, Psychiatric Quart., **19**:465–77, 1945.
33. Benton, A. L. Mental development of prematurely born, Am. J. Orthopsychiat., **10**:719–46, 1940.
34. Berger, H. Ueber das Elektrenkephalogram des Menschen. I, Arch. f. Psychiat., **87**:527–70, 1929.
35. Berry, R. J. A. A cerebral atlas. London: Oxford University Press, 1938. Pp. xxvi+425.
36. Bianchi, L. The mechanism of the brain. Edinburgh: E. & S. Livingstone, 1922. Pp. xx+348.
37. Binet, A. Les idées modernes sur les enfants. Paris: Ernest Flammarion, 1909. Pp. 346.
38. Binet, A., and Simon, T. The development of intelligence in children. Baltimore: Williams & Wilkins, 1916. Pp. 336.
39. Bishop, G. H. Electrical responses accompanying activity of the optic pathway, Arch. Ophth., **14**:992–1019, 1935.
40. Bishop, G. H., and O'Leary, J. L. Response of the optic cortex of the cat, Proc. Soc. Exper. Biol. & Med., **38**:532–35, 1938.
41. Bishop, G. H., and Bartley, S. H. Activity in the optic system following stimulation by brief flashes of light, Proc. Soc. Exper. Biol. & Med., **46**:557–58, 1941.
42. Bleckwenn, W. J. The use of sodium amytal in catatonia, A. Research Nerv. & Ment. Dis., Proc., **10**:224–35, 1931.
43. Bleuler, E. Textbook of psychiatry. London: Allen & Unwin, 1923. Pp. xviii+635.
44. Bolles, M. M. The basis of pertinence: a study of the test performance of aments, dements and normal children of the same mental age, Arch. Psychol., No. 212, 1–51, 1937.
45. Bonin, G. von. On the size of man's brain as indicated by skull capacity, J. Comp. Neurol., **59**:1–28, 1934.
46. ———. On encephalometry: a preliminary study of the brain of man, chimpanzee and macaque, ibid., **75**:287–314, 1941.
47. Boring, E. G. A history of experimental psychology. New York: Century Co., 1929. Pp. xvi+699.
48. Bouman, L., and Grunbaum, A. A. Experimentelle-psychologische Untersuchun-

gen zur Aphasie und Paraphasie, Ztschr. f. d. ges. Neurol. u. Psychiat., **96**:481–538, 1925.

49. Braak, J. W. G. ter. Untersuchungen über optokinetischen Nystagmus, Arch. néerl. de physiol., **21**:310–75, 1936.
50. Breasted, J. H. The Edwin Smith surgical papyrus. Chicago: University of Chicago Press, 1930. Pp. xxiv+8 pls.+596.
51. Brecher, A. Die Verschmelzungsgrenze von Lichtreizen beim Affen, Ztschr. f. vergl. Physiol., **22**:539–47, 1935.
52. Breslaw, B.; Barrera, S. E.; and Warden, C. J. The effect of removal of the post-central convolution of the macacus rhesus monkey upon the delayed response, J. Comp. Psychol., **18**:207–26, 1934.
53. Brickner, R. M. The intellectual functions of the frontal lobes. New York: Macmillan Co., 1936. Pp. xvi+354.
54. ———. Bilateral frontal lobectomy: follow-up report of a case, Arch. Neurol. & Psychiat., **41**:580–85, 1939.
55. Broca, P. Remarques sur le siège de la faculté du langage articulé, suivies d'une observation d'aphémie (perte de la parole), Bull. Soc. anat., 2d ser., **36**:330–57, 1861.
56. Brodmann, K. Vergleichende Lokalisationslehre der Grosshirnrinde. Leipzig, J. A. Barth. Pp. x+324.
57. Brosin, H. W., and Halstead, W. C. Rorschach performances of patients with cerebral injury. Presented on the program of the Midwestern Psychological Assoc., May, 1938.
58. Brouwer, B. Projection of macula to area striata: case of thrombotic softening in left occipital lobe with right-sided hemianopia and preservation of central vision, J. Psychol. & Neurol., **40**:147–59, 1930.
59. Brouwer, B., and Zeeman, W. P. C. The projection of the retina in the primary optic neurone in monkeys, Brain, **49**:1–35, 1926.
60. Brown, C. W. Subcortical mechanisms in learning. I. The functional significance of subcortical nuclei in certain simple learning tasks, with a description of a program for further experimental work, Psychol. Rev., **42**:307–34, 1935.
61. Brown, C. W., and Ghiselli, E. E. Subcortical mechanisms in learning. II. The maze, J. Comp. Psychol., **26**:27–44, 1938.
62. ———. Subcortical mechanisms in learning. VI. Pattern vision discrimination, ibid., pp. 287–300.
63. Brown, C. W., and Henry, E. M. The central nervous system for emotional responses. II. A technique for destroying the deeper nuclear regions within the cerebrum with a minimal destruction of the intervening cortex, Proc. Nat. Acad. Sc., **20**:310–15, 1934.
64. Brozek, J., and Keys, A. Changes in flicker-fusion frequency with age, J. Consulting Psychol., **9**:87–90, 1945.
65. Brücke, E. Ueber den Nutzeffect intermittirende Netzhautreizungen, Sitzungsb. Akad. Wiss. Wien, **49**, No. 1, 128–53, 1864.
66. Brunswik, E., and Reiter, L. Eindruckscharactere schematisierter Gesichter, Ztschr. f. Psychol., **142**:67–134, 1937.
67. Bryan, A. H., and Ricketts, H. T. Effect in man of chronic intermittent anoxia on glucose tolerance and the urinary excretion of sodium, potassium, chloride, phosphorus, nitrogen, and 17-ketosteroids, J. Clin. Endocrinol., **4**:450–64, 1944.
68. Bucy, P. C. Frontal lobe of primates: relation of cytoarchitecture to functional activity, Arch. Neurol. & Psychiat., **33**:546–57, 1935.
69. ———. Neurosurgical treatment of certain abnormal mental states, J.A.M.A., **117**:517–27, 1941.

70. BUNGE, E. Verlauf der Dunkeladaptation bei Sauerstoffmangel, Arch. f. Augenh., **110**:189–97, 1936–37.
71. BURT, C.; JONES, E.; MILLER, E.; and MOODIS, W. How the mind works. New York: Appleton-Century, 1934. Pp. 336.
72. CAMERON, N. Cerebral destruction in its relation to maze learning, Psychol. Monog., No. 39, Part I, pp. 1–68, 1928.
73. ———. A study of thinking in senile deterioration and schizophrenic disorganization, Am. J. Psychol., **51**:650–65, 1938.
74. ———. Deterioration and regression in schizophrenic thinking, J. Abnorm. & Social Psychol., **34**:265–70, 1939.
75. ———. Schizophrenic thinking in a problem-solving situation, J. Ment. Sc., **85**: 1–24, 1939.
76. CAMPBELL, C. M. Adolf Meyer, Arch. Neurol. & Psychiat., **37**:715–31, 1937.
77. CARLSON, H. B.; GELLHORN, E., and DARROW, C. W. Representation of the sympathetic and parasympathetic nervous systems in the forebrain of the cat, Arch. Neurol. & Psychiat., **45**:105–16, 1941.
78. CASE, T. J. Electrical aspects of the epilepsies, Dis. Nerv. System, **2**:3–7, 1941.
79. ———. Alpha waves in relation to structures involved in vision. In: KLÜVER, H. (ed.), Biol. Symposia, **7**:107–16, 1942.
80. CASE, T. J., and BUCY, P. C. Localization of cerebral lesions by electroencephalography, J. Neurophysiol., **1**:245–61, 1938.
81. CATE, J. TEN; WALTER, W. G.; and KOOPMAN, L. J. Electroencephalography after removal of the occipital cortex, Arch. néerl. de physiol., **24**:153–68, 1939.
82. CHASE, A. M., and HAGAN, W. H. The photochemical and thermal reactions of visual purple in absence of oxygen, J. Cell. & Comp. Physiol., **21**:65–75, 1943.
83. CLARK, W. E. LE GROS. Anatomical basis of color vision, Nature, **146**:558–59, 1940.
84. ———. Observations on the association fibre system of the visual cortex and the central representation of the retina, J. Anat., **75**:225–35, 1941.
85. CLOWARD, R. B. War injuries to the head, J.A.M.A., **118**:267–70, 1942.
86. COHN, R. Prefrontal lobotomy: electroencephalographic study of focal brain injury, Arch. Neurol. & Psychiat., **53**:283–88, 1945.
87. COHN, H. A., and PAPEZ, J. W. A comparative study of the visuosensory or striate area in the two hemispheres of the human brain, Am. J. Phys. Anthropol., **14**: 405–15, 1930.
88. CORDS, R. Optisch-motorisches Feld und optisch-motorische Bahn, Arch. f. Ophth., **117**:58–114, 1926.
89. COURVILLE, C. B. Coup-contrecoup mechanism of craniocerebral injuries: some observations, Arch. Surg., **45**:19–43, 1942.
90. CROTHERS, B., and LORD, E. Appraisal of intellectual and physical factors after cerebral damage in children, Am. J. Psychiat., **94**:1077–88, 1938.
91. CROZIER, W. J., and WOLF, E. The simplest flicker threshold contour for the zebra finch, J. Gen. Physiol., **24**:625–33, 1941.
92. ———. Theory and measurement of visual mechanisms. IV. Critical intensities for visual flicker, monocular and binocular, ibid., pp. 505–34.
93. ———. Theory and measurement of visual mechanisms. VI. Wavelength and flash duration in flicker, ibid., **25**:89–110, 1941.
94. ———. Theory and measurement of visual mechanisms. VII. The flicker response function outside the fovea, ibid., pp. 293–308.
95. ———. Theory and measurement of visual mechanisms. VIII. The form of the flicker contour, ibid., **25**:369–79, 1942.

96. CULLER, E., and METTLER, F. A. Conditioned behavior in a decorticate dog, J. Comp. Psychol., **18**:291–303, 1934.
97. DANDY, W. E. Contributions to brain surgery, Ann. Surg., **4**:89–93, 1925.
98. ———. Removal of right cerebral hemisphere for certain tumors with hemiplegia: preliminary report, J.A.M.A., **90**:823–25, 1928.
99. ———. Physiologic study following extirpation of right cerebral hemisphere in man, Bull. Johns Hopkins Hosp., **53**:31–51, 1933.
100. DARROW, C. W. Emotion as relative functional decortication: the role of conflict, Psychol. Rev., **42**:566–78, 1935.
101. DARROW, C. W.; GREEN, J. R.; DAVIS, E. W.; and GAROL, H. W. Parasympathetic regulation of high potential in the electroencephalogram, J. Neurophysiol., **7**:217–26, 1944.
102. DARROW, C. W., and PATHMAN, J. H. The role of blood pressure in electroencephalographic changes during over-ventilation, Federation Proc., **2**:9, 1943.
103. ———. Relation of heart rate to slow waves in the electroencephalogram during over-ventilation, Am. J. Physiol., **140**:583–88, 1944.
104. DAVIS, P. A. Electroencephalographic studies on three cases of frontal lobotomy, Psychosom. Med., **3**:38–50, 1941.
105. DAX, E. C., and RADLEY SMITH, E. J. Prefrontal leukotomy: early effects on disturbed patients with mental illness of long duration, J. Ment. Sc., **89**:182–85, 1943.
106. DEARBORN, W. F.; ANDERSON, J. E.; and CHRISTIANSEN, A. O. Form board and construction tests of mental ability, J. Ed. Psychol., **7**:445–58, 1916.
107. DELMAS, M. P. Lobe frontal et equilibre, J. belge de neurol. et de psychiat., **35**:458–70, 1935.
108. DIEFENDORF, A. R. Clinical psychiatry. New York: Macmillan Co., 1923. Pp. xvii+562.
109. DIKSHIT, B. B. Action of acetylcholine on the brain and its occurrence therein, J. Physiol., **80**:409–21, 1934.
110. DIVEN, K. Dandy's radical extirpations of brain tissue in man, Am. J. Psychol., **46**:500–503, 1934.
111. DOBRINER, K., and RHOADS, C. P. The porphyrins in health and disease, Physiol. Rev., **20**:416–68, 1940.
112. DOLIN, A. D.; MINKER-BOGDANOVA, E. T.; and POVARINSKY, J. A. The significance of the cerebral cortex in metabolism disorders, Arkh. Biol. Nauk., **36**:65–78, 1934.
113. DRAGSTEDT, L., and OWENS, F. M., JR. Supra-diaphragmatic section of the vagus nerves in treatment of duodenal ulcers, Proc. Soc. Exper. Biol. & Med., **53**:152–54, 1943.
114. DUNLAP, K. Psychologic observations and methods. IV. Medical studies in aviation, J.A.M.A., **71**:1382–1400, 1918.
115. DURUP, G., and FESSARD, A. L'électrencephalogramme de l'homme: observations psycho-physiologiques relative a l'action des stimuli visuels et auditifs, Ann. psychol., **36**:1–35, 1935.
116. DUSSER DE BARENNE, J. G. Central levels of sensory integration, Arch. Neurol. & Psychiat., **34**:768–76, 1935.
117. EBAUGH, F. G., and BROSIN, H. W. Traumatic psychoses, Ann. Int. Med., **18**:666–96, 1943.
118. EBBINGHAUS, H. Über eine neue Methode zur Prüfung geistiger Fähigkeiten und ihre Anwendung bei Schulkindern, Ztschr. f. Psychol., **13**:401–59, 1897.
119. ECONOMO, C. VON, and KOSKINAS, G. N. Die Cytoarchitektonik der Hirnrinde

des erwachsenen Menschen. Berlin and Vienna: Julius Springer, 1925. Pp. xxxix+810.

120. ELSBERG, C. A. The sense of smell: the value of quantitative olfactory tests for the localization of supratentorial tumors of the brain: a preliminary report, J. Nerv. & Ment. Dis., **83**:319–28, 1936.

121. ENGEL, G. L.; WEBB, J. P.; FERRIS, E. B.; ROMANO, J.; RYDER, H.; and BLANKENHORN, M. A. A migraine-like syndrome complicating decompression sickness, War Med., **5**:304–14, 1944.

122. EPSTEIN, M. A., and MORGAN, C. T. Cortical localization of symbolic processes in the rat. III. Impairment of anticipatory functions in prefrontal lobectomy in rats, J. Exper. Psychol., **32**:453–63, 1943.

123. ERICKSEN, S. C. The relative effect of a cerebral lesion on learning, retention and transfer, J. Comp. Psychol., **27**:373–91, 1939.

124. EVANS, J. N., and MCFARLAND, R. A. The effects of oxygen deprivation on the central visual field, Am. J. Ophth., **21**:968–80, 1938.

125. FEARING, F. Reflex action: a study in the history of physiological psychology. Baltimore: Williams & Wilkins, 1930. Pp. xiii+350.

126. FERENCZI, S. Further contributions to the theory and technique of psychoanalysis. New York: Boni & Liveright, 1927. Pp. xiv+458.

127. FERRIER, D. The functions of the brain. London: Smith, Elder & Co., 1886. Pp. xxiii+498.

128. FEUCHTWANGER, E. Die Funktionen des Stirnhirn, ihre Pathologie und Psychologie, Monog. a.d. Ges. d. Neurol. u. Psychiat., No. 38, 1923. Pp. xxxviii+194.

129. FINAN, J. L. Effects of frontal lobe lesions on the temporally organized behavior in monkeys, J. Neurophysiol., **2**:208–26, 1939.

130. FISCHER, F. P., and JONGBLOED, J. Untersuchungen über die Dunkeladaptation bei herabgesetztem Sauerstoffdruck der Atmungsluft, Arch. f. Augenh., **109**:452–56, 1935–36.

131. FISCHER, M. H. Elektrobiologische Erscheinungen an der Hirnrinde. I, Arch. f. d. ges. Physiol., **230**:161–78, 1932.

132. ———. Elektrobiologische Erscheinungen an der Hirnrinde bei Belichtung eines Auges. II, ibid., **233**:738–53, 1934.

133. FISHER, R. A. Statistical methods for research workers. London: Oliver & Boyd, 1928. Pp. xi+269.

134. ———. The design of experiments. London: Oliver & Boyd, 1935. Pp. xi+252.

135. FLACK, M. Test for flying efficiency and flying strain, Brit. Privy Council, Med. Research Council, Special Rept. Series, No. 53, pp. 93–140, 1920.

136. FLECHSIG, P. Die Körperlichen Grundlagen der Geistestörungen. Leipzig: Veit &. Co., 1882. Pp. 36.

137. ———. Gehirn und Seele. Leipzig: Veit & Co., 1896. Pp. 112.

138. ———. Die Lokalisation der Geistigen Vorgänge. Leipzig: Veit & Co., 1896. Pp. 88.

139. ———. Die Lokalisation der geistigen Funktionen, Zentralbl. f. d. ges. Neurol. u. Psychiat., **31**:61–62, 1922–23.

140. FLEMING, G. W. T. H. Prefrontal leukotomy: preliminary remarks, J. Ment. Sc., **88**:282–84, 1942.

141. ———. Prefrontal leukotomy: recent progress in psychiatry, ibid., **90**:486–500, 1944.

142. FLEMING, G. W. T. H., and MCKISSOCK, W. Prefrontal leukotomy: further contribution, Lancet, **1**:361–62, 1943.

143. FLOURENS, M. J. P. Recherches experimentales sur les propriétés et les fonctions du système nerveux. Paris: J. B. Baillière, 1842. Pp. xxviii+516.

144. FORSTER, E. Die Psychischen Störungen der Hirnverletzten, Monatschr. f. Psychiat. u. Neurol., 46:61–105, 1919.

145. FORTUYN, A. E. B. D. Cortical cell-laminations of the brains of some rodents, Arch. Neurol. & Psychiat., 6:221–354, 1914.

146. FOX, J. C. Disorders of optic nystagmus due to cerebral tumors, Arch. Neurol. & Psychiat., 28:1007–29, 1932.

147. FOX, J. C., and HOLMES, G. Optic nystagmus and its value in the localization of cerebral lesions, Brain, 49:333–72, 1926.

148. FOX, J. C., and GERMAN, W. J. Macular vision following cerebral resection, Arch. Neurol. & Psychiat., 35:808–26, 1936.

149. FRANKEN, A. Bilderkombinationen: Ein Beitrag zum Problem der Intelligenzprüfung, Ztschr. f. ang. Psychol., 12:173–329, 1917.

150. FRANZ, S. I. On the functions of the cerebrum: the frontal lobes, Monog. No. 2, Arch. Psychol. New York: Science Press, 1907. Pp. 64.

151. FRANZ, S. I., and GORDON, K. Psychology. New York: McGraw-Hill Book Co., 1933. Pp. xvii+494.

152. FREEMAN, W., and WATTS, J. W. Interpretation of functions of frontal lobe based upon observations in 48 cases of prefrontal lobotomy, Yale J. Biol. & Med., 11: 527–39, 1939.

153. ———. Frontal lobes and consciousness of self (results of prefrontal lobotomy), Psychosom. Med., 3:111–19, 1941.

154. ———. Psychosurgery: intelligence, emotion, and social behavior following prefrontal lobotomy for mental disorders. Springfield, Ill: Charles C. Thomas, 1942. Pp. xii+337.

155. ———. Psychosurgery: evaluation of 200 cases (of prefrontal lobotomy) over 7 years, J. Ment. Sc., 90:532–37, 1944.

156. FREUD, S. The ego and the id. London: Hogarth Press, 1927. Pp. 88.

157. ———. The future of an illusion. London: Hogarth Press, 1934. Pp. 98.

158. ———. The basic writings of Sigmund Freud. New York: Modern Library, 1938, Pp. vi+1001.

159. FRITSCH, G., and HITZIG, E. Ueber die electrische Erregbarkeit des Grosshirn, Arch. f. Anat. u. Physiol., 37:300–332, 1870.

160. FULTON, J. F. Selected readings in the history of physiology. Springfield, Ill.: Charles C. Thomas, 1930. Pp. xx+317.

161. FULTON, J. F.; LIDDELL, E. G. T.; and RIOCH, D. M. Relation of the cerebrum to the cerebellum. I. Cerebellar tremor in the cat and its absence after removal of the cerebral hemispheres, Arch. Neurol. & Psychiat., 28:542–67, 1932.

162. GELB, A., and GOLDSTEIN, K. Ueber Farbennamenamnesie, Psychol. Forsch., 6:127–86, 1925.

163. GELLHORN, E. Anoxia in relation to the visual system. In: KLÜVER, H. (ed.), Biol. Symposia, 7:73–85, 1942.

164. GERMAN, W. J. Epileptogenic cortical scars: results of surgical removal, Arch. Neurol. & Psychiat., 41:73–82, 1939.

165. GERMAN, W. J., and FOX, J. C. Observations following unilateral lobectomies, A. Research Nerv. & Ment. Dis., Proc., 13:378–434, 1932.

166. GIBBS, F. A.; GIBBS, E. L.; and LENNOX, W. G. The cerebral blood flow during sleep in man, Brain, 58:44–49, 1935.

167. GIRDEN, E. The role of the auditory area of the cortex, Am. J. Psychol., 53:371–83, 1940.

168. GOLDMAN, G.; SEGAL, J.; and SEGALIS, M. L'action d'une excitation intermittente sur le rythme de Berger, Compt. rend. Soc. de Biol., 127:1217–20, 1938.

169. GOLDMANN, H., and SCHUBERT, G. Untersuchungen über das Gesichtsfeld bei

herabgesetztem Sauerstoffdruck der Atmungsluft, Arch. Augenh., **107**:216–37, 1933.
170. GOLDSTEIN, K. Die Lokalisation in der Grosshirnrinde, Handb. d. norm. u. path. Physiol., **10**:600–842, 1927.
171. ———. Der Aufbau des Organismus: Einfuhrung in die Biologie unter besonderes Berucksichtingung der Erfahrungen am kranken Menschen. Hoog: Martinus Nijhoff, 1934. Pp. 363.
172. ———. The function of the cerebellum from a clinical standpoint, J. Nerv. & Ment. Dis., **83**:1–12, 1936.
173. ———. The significance of the frontal lobes for mental performances, J. Neurol. & Psychopathol., **17**:27–40, 1936.
174. ———. Clinical and theoretic aspects of lesions of the frontal lobes, Arch. Neurol. & Psychiat., **41**:865–67, 1939.
175. ———. Human nature. Cambridge, Mass.: Harvard University Press, 1940. Pp. x+258.
176. ———. After-effects of brain injuries in war. New York: Grune & Stratton, 1942. Pp. 244.
177. ———. The two ways of adjustment of the organism to cerebral defects, J. Mt. Sinai Hosp., **9**:504–13, 1942.
178. ———. Brain concussion: evaluation of the after-effects by special tests, Dis. Nerv. System, **4**:3–12, 1943.
179. ———. The mental changes due to frontal lobe damage, J. Psychol., **17**:187–208, 1944.
180. GOLDSTEIN, K., and ROSENTHAL-VEIT, O. Uber akustische Lokalisation und deren Beeinfluzbarkeit durch andere Sinnesreize, Psychol. Forsch., **8**:318–35, 1926.
181. GOLDSTEIN, K., and SCHEERER, M. Abstract and concrete behavior: an experimental study with special mental tests, Psychol. Monog., No. 239, 1941. Pp. 151.
182. GOLTZ, F. Uber die Verrichtungen des Grosshirns, Arch. f. d. ges. Physiol., **26**:1–49, 1881.
183. GRINKER, R. R., and SEROTA, H. M. Electroencephalographic studies of cortico-hypothalamic relations in schizophrenia, Am. J. Psychiat., **98**:385–92, 1941.
184. GRÜNTHAL, E. Ueber die Erkennung der traumatischen Hirnverletzung. Berlin: Karger, 1936. Pp. 116.
185. GUILFORD, J. P. Psychometric methods. New York: McGraw-Hill Book Co., 1936. Pp. xvi+566.
186. HALL, J. K. (ed.). One hundred years of American psychiatry. New York: Columbia University Press, 1944. Pp. xxiv+649.
187. HALSTEAD, W. C. The effects of cerebellar lesions upon the habituation of post-rotational nystagmus. Summaries of Doctoral Dissertations, Northwestern University, **3**:279–83, 1935.
188. ———. The effects of cerebellar lesions upon the habituation of post-rotational nystagmus, Comp. Psychol. Monog., **12**, No. 56, 1935. Pp. 130.
189. ———. A method for the quantitative recording of eye movements, J. Psychol., **6**:177–80, 1938.
190. ———. Behavioral effects of lesions of the frontal lobe in man, Arch. Neurol. & Psychiat., **42**:780–83, 1939.
191. ——— Preliminary analysis of grouping behavior in patients with cerebral injury by the method of equivalent and non-equivalent stimuli, Am. J. Psychiat., **96**:1263–94, 1940.
192. ———. A note on the Bartley effect in the estimation of equivalent brightness, J. Exper. Psychol., **28**:524–28, 1941.

193. ———. Brain injuries and the higher levels of consciousness. Chap. xx in: Trauma of the central nervous system. A. Research Nerv. & Ment. Dis., Proc., 1943. Baltimore: Williams & Wilkins, 1945.

194. Halstead, W. C., *et al.* The effects of chronic intermittent anoxia: neuropsychological aspects, Committee on Aviation Medicine Rept. 159, 1943.

195. Halstead, W. C. Chronic intermittent anoxia and the dynamic visual field, J. Psychol., **20**:49–56, 1945.

196. ———. Chronic intermittent anoxia and impairment of peripheral vision, Science, **101**:615–16, 1945.

197. ———. Correction on "chronic intermittent anoxia," ibid., **102**:159, 1945.

198. ———. Localization of neuropsychological functions in the prefrontal lobes, Federation Proc., **4**:13–14, 1945.

199. ———. A power factor (P) in general intelligence: the effect of brain injuries, J. Psychol., **20**:57–64, 1945.

200. ———. The principle of blended samples in validating behavioral indicators, Federation Proc., **4**: 12–13, 1945.

201. ———. A technique for charting neurosurgical lesions, J. Neurosurg., **3**:453–57, 1946.

202. Halstead, W. C.; Carmichael, H. T.; and Bucy, P. C. Prefrontal lobotomy: a preliminary appraisal of the behavioral results, Am. J. Psychiat., **103**:217–28, 1946.

203. Halstead, W. C.; Knox, G. W.; and Walker, A. E. Modification of cortical activity by means of intermittent photic stimulation in the monkey, J. Neurophysiol., **5**:349–56, 1942.

204. Halstead, W. C.; Knox, G. W.; Woolf, J. I.; and Walker, A. E. Effects of intensity and wave length on driving cortical activity in monkeys, J. Neurophysiol., **5**:483–86, 1942.

205. Halstead, W. C., and Settlage, P. H. Grouping behavior of normal persons and of persons with lesions of the brain, Arch. Neurol. & Psychiat., **49**:489–506, 1943.

206. Halstead, W. C.; Walker, A. E.; and Bucy, P. C. Sparing and nonsparing of "macular" vision associated with occipital lobectomy in man, Arch. Ophth., **24**: 948–62, 1940.

207. Halstead, W. C., and Yacorzynski, G. K. A jumping method for establishing differential responses in pigeons, J. Genet. Psychol., **52**:227–31, 1938.

208. Halstead, W. C.; Yacorzynski, G. K.; and Fearing, F. Further evidence of cerebellar influence in the habituation of after-nystagmus in pigeons, Am. J. Physiol., **120**:350–55, 1937.

209. Hanfmann, E., and Kasanin, J. A method for the study of concept formation, J. Psychol., **3**:521–40, 1937.

210. Hanfmann, E.; Rickers-Ovsiankina, M.; and Goldstein, K. Case Lanuti: extreme concretization of behavior due to damage of the brain cortex, Psychol. Monog., **57**, No. 4, 1–72, 1944.

211. Harbinson, M. R. An investigation of deterioration of "general intelligence" or "G" in psychotic patients, Brit. J. M. Psychol., **16**:146–48, 1936.

212. Harrower, M. R. Changes in figure-ground perception in patients with cortical lesions, Brit. J. Psychol., **30**:48–51, 1939.

213. Harrower-Erickson, M. R. Personality changes accompanying cerebral lesions. I. Rorschach studies of patients with cerebral tumors, Arch. Neurol. & Psychiat., **43**:859–90, 1940.

214. Hayman, M. Two-minute clinical test for measurement of intellectual impairment, Arch. Neurol. & Psychiat., **47**:454–64, 1942.

215. HEAD, H. Aphasia and kindred disorders of speech. 2 vols. New York: Macmillan Co., 1926. Pp. xiv+549; xxxiii+430.
216. HEBB, D. O. Intelligence in man after large removals of cerebral tissue: report of four left frontal lobe cases, J. Gen. Psychol., **21**:73–87, 1939.
217. ———. Intelligence in man after large removals of cerebral tissue: defects following right temporal lobectomy, ibid., pp. 437–46.
218. ———. Human intelligence after removal of cerebral tissue from the right frontal lobe, ibid., **25**:257–65, 1941.
219. ———. The effect of early and late brain injury upon test scores, and the nature of normal adult intelligence, Proc. Am. Phil. Soc., **85**:275–92, 1942.
220. HEBB, D. O., and PENFIELD, W. Human behavior after extensive bilateral removal from the frontal lobes, Arch. Neurol. & Psychiat., **44**:421–38, 1940.
221. HECHT, S., *et al.* The effect of anoxia on visual contrast discrimination, Committee on Aviation Medicine Rept. 184, 1943.
222. HECHT, S., and VERRIJP, C. D. Intermittent stimulation by light. III. The relation between intensity and critical fusion frequency for different retinal locations, J. Gen. Physiol., **17**:251–68, 1933.
223. HECHT, S., and WOLF, E. Intermittent stimulation by light. I. The validity of Talbot's law for *Mya*, J. Gen. Physiol., **15**:369–89, 1932.
224. HEILBRUNN, G., and HLETKO, P. Disappointing results with bilateral prefrontal lobotomy in chronic schizophrenia, Am. J. Psychiat., **99**:569–70, 1943.
225. HEMPHILL, R. E. Return of virility after prefrontal leukotomy (in case of obsessional neurosis); with enlargement of gonads, Lancet, **2**:345–46, 1944.
226. HEMPHILL, R. E.; MACLEOD, L. D.; and REISS, M. Changes in output of 17-ketosteroids after prefrontal leukotomy, J. Ment. Sc., **88**:554–58, 1942.
227. HERRICK, C. J. Neurological foundations of animal behavior. New York: Henry Holt & Co., 1924. Pp. xii+334.
228. ———. Morphogenesis of the brain, J. Morphol., **54**:233–58, 1933.
229. HESDORFFER, M. B., and SCAMMON, R. E. Growth of human nervous system—growth of cerebral surface, Proc. Soc. Exper. Biol. & Med., **33**:415–18, 1935.
230. HITZIG, E. Untersuchungen über das Gehirn. Berlin: A. Hirschwald, 1874. Pp. xiii+276.
231. ———. Zur Physiologie des Grosshirns, Arch. f. Psychiat., **15**:270–75, 1884.
232. ———. Physiologische und klinische Untersuchungen über das Gehirn. Gesammelte Abhandlungen. Vol. I: Untersuchungen über das Gehirn. Vol. II: Alte und neue Untersuchungen über das Gehirn. Berlin: A. Hirschwald, 1904. Pp. xvii+618.
233. HOLBOURN, A. H. S. Mechanics of head injuries, Lancet, **2**:438–41, 1943.
234. HOLZINGER, K. J. Statistical résumé of the Spearman two-factor theory. Chicago: University of Chicago Press, 1930.
235. ———. Preliminary report on Spearman-Holzinger unitary trait study, Nos. 1–8. Chicago: University of Chicago Press, 1935.
236. HOLZINGER, K. J., and HARMON, H. H. Relationships between factors obtained from certain analyses, J. Ed. Psychol., **28**:321–45, 1937.
237. ———. Factor analysis: a synthesis of factorial methods. Chicago: University of Chicago Press, 1941. Pp. xii+417.
238. HUNT, T. Psychological testing of psychiatric patients undergoing prefrontal lobotomy, Psychol. Bull., **37**:566, 1940.
239. HUTTON, E. L. Investigation of personality in patients treated by prefrontal leukotomy, J. Ment. Sc., **88**:275–81, 1942.
240. IVY, A. C. Experimental studies on the brain. I. Comparative study of the rela-

tion of the cerebral cortex to vestibular nystagmus, J. Comp. Neurol., 31:1–15, 1919.

241. Jacobsen, C. F. Studies of cerebral function in primates. I. The function of the frontal association areas in monkeys, Comp. Psychol. Monog., 13:1–60, 1936.

242. Jacobsen, C. F., and Elder, J. H. Studies of cerebral function in primates. II. The effect of temporal lobe lesions on delayed response in monkeys, Comp. Psychol. Monog., 13:61–65, 1936.

243. Jacobsen, C. F., and Nissen, H. W. Studies in the cerebral function in primates. IV. The effects of frontal lobe lesions on the delayed alternation habit in monkeys, J. Comp. Psychol., 23:101–12, 1937.

244. Jacobsen, C. F.; Taylor, F. V.; and Haslerud, G. M. Restitution of function after cortical injury in monkeys, Am. J. Physiol., 116:85–86, 1936.

245. Jacobsen, C. F.; Wolfe, J. B.; and Jackson, T. A. An experimental analysis of the functions of the frontal association areas in primates, J. Nerv. & Ment. Dis., 82: 1–14, 1935.

246. Janet, P. The major symptoms of hysteria. New York: Macmillan Co., 1907. Pp. 345.

247. Jasper, H. H. Electrical signs of cortical activity, Psychol. Bull., 34:411–81, 1937.

248. Jefferson, G. Left frontal lobectomy, Proc. Roy. Soc. Med., 30:851–53, 1937.

249. ———. Removal of right or left frontal lobes in man, Brit. M. J., 2:199–206, 1937.

250. Jung, C. G. Psychological types. New York: Harcourt, Brace & Co., 1923. Pp. 654.

251. Kappers, C. U. A.; Huber, G. C.; and Crosby, E. C. The comparative anatomy of the nervous system of vertebrates including man. 2 vols. New York: Macmillan Co., 1936. Pp. xi+980.

252. Kasanin, J., and Hanfmann, E. Disturbances in concept formation in schizophrenia, Arch. Neurol. & Psychiat., 40:1276–82, 1938.

253. ———. An experimental study of concept formation in schizophrenia. I. Quantitative analysis of the results, Am. J. Psychiat., 95:35–48, 1938.

254. Kelley, T. L. Crossroads in the mind of man: a study of differentiable mental abilities. Stanford University, Calif.: Stanford University Press, 1928. Pp. vii+238.

255. Kennard, M. A. Alteration in response to visual stimuli following lesions of the frontal lobe in monkey, Arch. Neurol. & Psychiat., 41:1153–65, 1939.

256. ———. Autonomic functions. Chap. xi in: Bucy, P. C. (ed.), The precentral motor cortex. Urbana, Ill.: University of Illinois Press, 1944.

257. Kennard, M. A., and Nims, L. F. Changes in electroencephalograms appearing coincident with growth in infant monkeys, Am. J. Physiol., 133:349–50, 1941.

258. ———. Changes in normal E.E.G. of *Macaca mulatta* with growth, J. Neurophysiol., 5:325–35, 1942.

259. ———. Effect on E.E.G. of lesions of cerebral cortex and basal ganglia in *Macaca mulatta*, ibid., pp. 335–48.

260. Kenyon, A. T.; Knowlton, K.; and Sandiford, I. Anabolic effects of the androgens and somatic growth in man, Ann. Int. Med., 20:632–54, 1944.

261. Kenyon, V. B., and Rapaport, D. The etiology of the psychosis of dementia paralytica with a preliminary report of the treatment of a case of the psychosis with metrazol, J. Nerv. & Ment. Dis., 94:147–59, 1941.

262. Kessler, M.; Hailman, H.; and Gellhorn, E. Studies on the effect of anoxic anoxia on the central nervous system, Am. J. Physiol., 140:291–98, 1943.

263. KIRK, S. A. Hemispheral equipotentiality and hemispheral cerebral dominance, Psychol. Bull., 32:743–44, 1935.

264. KISKER, G. W. Perceptual-motor patterns following bilateral prefrontal lobotomy, Arch. Neurol. & Psychiat., 50:691–96, 1943.

265. ———. Abstract and categorical behavior following therapeutic surgery, Psychosom. Med., 6:146–50, 1944.

266. KLEIST, K. Diagnostische und therapeutische Erfahrungen an Hirntumoren, Neurol. Centralbl., 38:207–8, 1919.

267. ———. Gehirnpathologische und localisatorische Ergebnisse: Die Storungen der Ichleistungen und ihre Lokalisation im Orbital-, Innen- und Zwischenhirn, Monatschr. f. Psychiat. u. Neurol., 79:338–50, 1931.

268. ———. Gehirnpathologie. Leipzig: J. A. Barth, 1934. Pp. 1408.

269. KLEITMAN, N., and CAMILLE, N. Studies on the physiology of sleep. VI. The behavior of decorticated dogs, Am. J. Physiol., 100:474–80, 1932.

270. KLÜVER, H. Visual disturbances after cerebral lesions, Psychol. Bull., 24:316–58, 1927.

271. ———. Behavior mechanisms in monkeys. Chicago: University of Chicago Press, 1933. Pp. xvii+387.

272. ———. An analysis of the effects of the removal of the occipital lobes in monkeys, J. Psychol., 2:49–61, 1936.

273. ———. The study of personality and the method of equivalent and nonequivalent stimuli, Char. & Pers., 5:91–112, 1936.

274. ———. Visual functions after removal of the occipital lobes, J. Psychol., 11:23–45, 1941.

275. ———. Functional significance of the geniculo-striate system. In: KLÜVER, H. (ed.), Biol. Symposia, 7:253–99, 1942.

276. KLÜVER, H., and BUCY, P. C. Preliminary analysis of the functions of the temporal lobes in monkeys, Arch. Neurol. & Psychiat., 42:979–1000, 1939.

277. KNOTT, J. R.; GIBBS, F. A.; and HENRY, C. E. Fourier transforms of the electroencephalogram during sleep, J. Exper. Psychol., 31:465–77, 1942.

278. KNOTT, J. R., and HENRY, C. E. The conditioning of the blocking of the alpha rhythm of the human electroencephalogram, J. Exper. Psychol., 28:134–44, 1941.

279. KNOX, G. W. Investigations of flicker and fusion. I. The effect of practice, under the influence of various attitudes, on the CFF, J. Gen. Psychol., 33:121–29, 1945

280. KÖHLER, W. Place of value in a world of facts. New York: Liveright, Pub. Corp. 1938. Pp. ix+418.

281. KORNMÜLLER, A. E. Architektonische Lokalisation bioelektrischer Erscheinungen auf der Grosshirnrinde. I. Untersuchungen am Kaninchen bei Augenbelichtung, J. f. Psychol. u. Neurol., 44:447–59, 1932.

282. ———. Die bioelektrischer Felder der Grosshirnrinde, Biol. Rev., 10:383–427, 1935.

283. KRAINER, L., and SUIVA, K. Anatomic projection and theory of double supply of macular fibers, Jahrb. f. Psychiat. u. Neurol., 53:35–44, 1936.

284. KRECHEVSKY, I. Brain mechanisms and variability. III. Limitations of the effect of cortical injury upon variability, J. Comp. Psychol., 23:351–63, 1937.

285. LANGWORTHY, O. R. The area frontalis of the cerebral cortex of the cat, its minute structure and physiological evidence of its control of the postural reflex, Bull. Johns Hopkins Hosp., 42:20–65, 1928.

286. LASHLEY, K. S. Brain mechanisms and intelligence. Chicago: University of Chicago Press, 1929. Pp. xiv+186.

287. ———. Integrative functions of the cerebral cortex, Physiol. Rev., **13**:1–42, 1933.

288. ———. The problem of cerebral organization in vision. In: Klüver, H. (ed.), Biol. Symposia, **7**:301–22, 1942.

289. Lashley, K. S.; McDonald, W. T.; and Peters, H. N. Studies of cerebral function in learning. X. The effects of dilation of the ventricles upon maze-learning, Am. J. Physiol., **104**:51–61, 1933.

290. Lashley, K. S., and Wade, M. The Pavlovian theory of generalization, Psychol. Rev., **53**:72–87, 1946.

291. Lashley, K. S., and Wiley, L. E. Studies of cerebral function in learning. IX. Mass action in relation to the number of elements in the problem to be learned, J. Comp. Neurol., **57**:3–49, 1933.

292. Liberson, W. T., and Seguin, C. A. Brain waves and heredity in mental diseases, Psychosom. Med., **7**:35–38, 1945.

293. Lidz, T. A study of the effect of right frontal lobectomy on intelligence and temperament, J. Neurol. & Psychiat., **2**:211–22, 1939.

294. Lidz, T.; Gay, J. R.; and Tietze, C. Intelligence in cerebral deficit states measured by Kohs Block Test, Arch. Neurol. & Psychiat., **48**:568–82, 1942.

295. Lindemann, E. Psychopathological effect of sodium amytal, Proc. Soc. Exper. Biol. & Med., **28**:864–67, 1931.

296. Loeb, J. Comparative physiology of the brain and comparative psychology. New York: G. P. Putnam's Sons, 1902. Pp. 309.

297. Loomis, A. L.; Harvey, E. N.; and Hobart, G. Electrical potentials of the human brain, J. Exper. Psychol., **19**:249–79, 1936.

298. Lorente de Nó, R. Studies on the structure of the cerebral cortex. II. Continuation of the study of the ammonic system, J. f. Psychol. u. Neurol., **46**:113–77, 1934–35.

299. Loucks, R. B. Efficacy of the rat's motor cortex in delayed alternation, J. Comp. Neurol., **53**:511–67, 1931.

300. McCulloch, W. S., and Dusser de Barenne, J. G. Extinction: local stimulatory inactivation within the motor cortex, Am. J. Physiol., **113**:97–98, 1935.

301. McDougall, W. Outline of abnormal psychology. New York: Charles Scribner's Sons, 1926. Pp. iv+426.

302. McFarland, R. A., and Evans, J. N. Alterations in dark adaptation under reduced oxygen tension, Am. J. Physiol., **127**:37–50, 1939.

303. Magoun, H. W.; Hare, W. K.; and Ranson, S. W. Electrical stimulation of the interior of the cerebellum in the monkey, Am. J. Physiol., **112**:329–39, 1935.

304. Maier, N. R. F. The effect of cerebral destruction on reasoning and learning in rats, J. Comp. Neurol., **54**:45–75, 1932.

305. ———. Cortical destruction in the posterior part of the brain and its effect on reasoning in rats, ibid., **56**:179–214, 1932.

306. ———. Qualitative differences in the problem solving of normal and partly decorticated rats, Mich. Acad. Sc. Arts & Letters, **24**:131–46, 1938.

307. Maison, G. L.; Settlage, P. H.; and Grether, W. F. Experimental study of macular representation in the monkey, Arch. Neurol. & Psychiat., **40**:981–84, 1938.

308. Malmo, R. B. Interference factors in delayed response in monkeys after removal of frontal lobes, J. Neurophysiol., **5**:295–308, 1942.

309. Malmo, R. B., and Finan, J. L. A comparative study of eight tests in the decompression chamber, Am. J. Psychol., **57**:389–405, 1944.

310. Marquis, D. G. Phylogenetic interpretation of the functions of the visual cortex, Arch. Neurol. & Psychiat., **33**:807–15, 1935.

311. Marquis, D. G., and Hilgard, E. R. Conditioned lid responses to light in dogs after removal of the visual cortex, J. Comp. Psychol., **22**:157–78, 1936.
312. ———. Conditioned responses to light in monkeys after removal of the occipital lobes, Brain, **60**:1–12, 1937.
313. Mettler, F. A. Cerebral function and cortical localization, J. Gen. Psychol., **13**:367–401, 1935.
314. Mettler, F. A.; Mettler, C. C.; and Culler, E. Effects of total removal of the cerebral cortex, Arch. Neurol. & Psychiat., **34**:1238–49, 1935.
315. Miles, W. R. The steady polarity potential of the human eye, Proc. Nat. Acad. Sc., **25**:25–36, 1939.
316. Minkowski, M. Zur Physiologie der Sehsphäre, Arch. f. d. ges. Physiol., **141**:171–327, 1911.
317. Monakow, C. von. Gehirnpathologie. Vienna: A. Hölder, 1905. Pp. xiv+1319.
318. ———. Die Lokalisation im Grosshirn und der Abbau der Funktion durch Kortikale Herde. Wiesbaden: Bergmann, 1914. Pp. 1033.
319. Moniz, E. Les possibilities de la chirugie dans le traitement de certaines psychoses, Lisboa méd., **13**:141–51, 1936.
320. ———. The first attempt at operative treatment of certain psychoses, Encéphale, **31**:1–29, 1936.
321. ———. Prefrontal leucotomy in treatment of mental disorders, Am. J. Psychiat. (Suppl.), **93**:1379–85, 1937.
322. Moore, T. V. The empirical determination of certain syndromes underlying praecox and manic depressive psychoses, Am. J. Psychiat., **9**:719–38, 1930.
323. ———. Studies in psychology and psychiatry. Vol. **3**, No. 3. Baltimore: Williams & Wilkins, 1933. Pp. 128.
324. ———. Formal causality and the analysis of mental life, J. Ed. Psychol., **25**:401–21, 1934.
325. Morgan, C. T., and Wood, W. M. Cortical localization of symbolic processes in the rat, J. Neurophysiol., **6**:173–80, 1943.
326. Mowrer, O. H.; Ruch, T. C.; and Miller, N. E. The corneo-retinal potential difference as the basis of the galvanometric method of recording eye movements, Am. J. Physiol., **114**:423–28, 1936.
327. Müller, R. H.; Garman, R. L.; and Droz, M. E. Experimental electronics. New York: Prentice-Hall, Inc., 1942. Pp. xv+330.
328. Munk, H. Ueber die Funktionen der Grosshirnrinde: gesamelte Mittheilungen mit Anmerkungen. Berlin: A. Hirschwald, 1890. Pp. 320.
329. Mussen, A. T. Experimental investigations on the cerebellum, Brain, **50**:313–49, 1927.
330. Nichols, I. C., and Hunt, J. M. A case of partial bilateral frontal lobectomy, Am. J. Psychiat., **96**:1063–83, 1940.
331. Page, I. H. Chemistry of the brain. Springfield, Ill.: Charles C. Thomas, 1937. Pp. xvii+444.
332. Patten, C. A., and Mathews, R. A. Cerebral birth conditions, with special reference to myelogeny, Arch. Neurol. & Psychiat., **34**:61–98, 1935.
333. Pavlov, I. P. Conditioned reflexes: an investigation of the physiological activity of the cerebral cortex. London: Oxford University Press, 1927. Pp. xvi+430.
334. Penfield, W. Epilepsy and surgical therapy, Arch. Neurol. & Psychiat., **36**:449–84, 1936.
335. ———. The cerebral cortex in man. I. The cerebral cortex and consciousness, ibid., **40**:417–42, 1938.
336. Penfield, W., and Boldrey, E. Somatic motor and sensory representation in

the cerebral cortex of man as studied by electrical stimulation, Brain, **60**:389–443, 1937.

337. PENFIELD, W., and ERICKSON, T. C. Epilepsy and cerebral localization. Springfield, Ill.: Charles C. Thomas, 1941. Pp. x+623.
338. PENFIELD, W., and EVANS, J. P. Functional defects produced by cerebral lobectomies, A. Research Nerv. & Ment. Dis., Proc., **13**:352–77, 1932.
339. ———. The frontal lobe in man: a clinical study of maximum removals, Brain, **58**:115–33, 1935.
340. PFEIFER, R. A. Pathologic findings in brain in case of bilateral hemianopia with preservation of macular vision, J. f. Psychol. u. Neurol., **40**:319–37, 1930.
341. PHILLIPS, G. Perception of flicker in lesions of the visual pathways, Brain, **56**:464–78, 1933.
342. PIOTROWSKI, Z. The Rorschach inkblot method in organic disturbances of the central nervous system, J. Nerv. & Ment. Dis., **86**:525–37, 1937.
343. POINCARÉ, H. The foundations of science. New York: Science Press, 1921. Pp. xi+553.
344. POPPELREUTER, W. Die psychischen Shädigungen durch Kopfschuss im Kriege 1914–1917. Leipzig: Voss, 1917. Pp. xvi+210.
345. PORTEUS, S. D. Medical applications of maze test (in prefrontal lobotomy), M. J. Australia, **31**:558–60, 1944.
346. PORTEUS, S. D., and KEPNER, R. D. Mental changes after bilateral prefrontal lobotomy, Genet. Psychol. Monog., **29**:4–115, 1944.
347. PUTNAM, T., and LIEBMAN, S. Cortical representation of the macula lutea with special reference to the theory of bilateral representation, Arch. Ophth., **28**:415–43, 1942.
348. RADEMAKER, G. G. J. Physiology of the cerebellum, Rev. neurol., **36**:337–67, 1930.
349. RORSCHACH, H. Psychodiagnostik, 2d ed. Bern: Hans Huber, 1932. Pp. 230.
350. ROTHMANN, E. Untersuchung eines Falles von umschreibener Hirnschädigung mit Störungen auf verschiedensten Leistungsgebieten, Schweiz. Arch. f. Neurol. u. Psychiat., **33**:3–51, 1933.
351. ROWE, S. N. Mental changes following the removal of the right cerebral hemisphere for brain tumor, Am. J. Psychiat., **94**:605–14, 1937.
352. RUBIN, M. A. The distribution of the alpha rhythm over the cerebral cortex of normal man, J. Neurophysiol., **1**:313–23, 1938.
353. RUCH, T. C., and SHENKIN, H. A. The relation of area 13 on orbital surface of frontal lobes to hyperactivity and hyperphagia in monkeys, J. Neurophysiol., **6**:349–60, 1943.
354. RYLANDER, G. Personality changes after operations on the frontal lobes. London: Oxford University Press, 1939. Pp. 327.
355. SAMUELS, M. R. Judgments of faces, Char. & Pers., **8**:18–27, 1939.
356. SCAMMON, R. E., and HESDORFFER, M. Growth of human nervous system. II. Indices of relation of cerebral volume to surface in developmental period, Proc. Soc. Exper. Biol. & Med., **33**:418–21, 1935.
357. SCHILDER, P. Psychic disturbances after head injuries, Am. J. Psychiat., **91**:155–88, 1934–35.
358. ———. Neuroses following head and brain injuries. In: BROCK, S. (ed.), Injuries of the skull, brain and spinal cord. Baltimore: Williams & Wilkins, 1940. Pp. xii+632.
359. SCHLIEPER, C. Der Farbensinn von Hippolyte, zugleich ein Beitrag zum Bewegungsehen der Krebse, Zool. Anz., Suppl. *b*, **2**:188–93, 1926.

360. ———. Ueber die Heligkeitsverteilung im Specktrum bei verschiedenen Inseckten, Ztschr. f. vergl. Physiol., **8**:281–88, 1928.

361. Seashore, C. E. Psychology of musical talent. Boston: Silver, Burdette & Co., 1919. Pp. xvi+288.

362. Seitz, C. P. The effects of anoxia on visual functions, Arch. Psychol., **36**:1–38, 1940.

363. Settlage, P. H. The effect of occipital lesions on visually guided behavior in the monkey. I. Influence of the lesions on final capacities in a variety of problem situations, J. Comp. Psychol., **27**:93–131, 1939.

364. Simonson, E., and Enzer, W. Measurement of fusion frequency of flicker as a test for fatigue of the central nervous system, J. Indust. Hyg. & Toxicol., **23**: 83–89, 1941.

365. Skinner, B. F. Behavior of organisms. New York: D. Appleton-Century Co., 1938. Pp. ix+457.

366. Smith, G. E. Cortical representation of macula, J. Anat., **64**:477–78, 1930.

367. Smith, K. U. Visual discrimination in the cat. VI. The relation between pattern vision and visual acuity and the optic projection centers of the nervous system, J. Genet. Psychol., **53**:251–72, 1938.

368. ———. The behavior of decorticate guinea pigs, J. Comp. Psychol., **27**:433–47, 1939.

369. ———. The neural centers concerned in the mediation of apparent movement vision, J. Exper. Psychol., **26**:443–66, 1940.

370. Smith, K. U., and Bridgman, M. The neural mechanisms of movement vision and optic nystagmus, J. Exper. Psychol., **33**:165–87, 1943.

371. Smith, K. U.; Kappauf, W. E.; and Bojar, S. The functions of the visual cortex in optic nystagmus at different velocities of movement in the visual field, J. Gen. Psychol., **22**:341–57, 1940.

372. Smith, K. U., and Warkentin, J. The central neural organization of optic functions related to minimum visible acuity, J. Genet. Psychol., **55**:177–95, 1939.

373. Smith, W. K. The representation of respiratory movements in the cerebral cortex, J. Neurophysiol., **1**:55–68, 1938.

374. Spatz, H. Uber die Bedeutung der basalen Rinde, Ztschr. f. d. ges. Neurol. u. Psychiat., **158**:208–32, 1937.

375. Spearman, C. The proof and measurement of association between two things, Am. J. Psychol., **15**:72–101, 1904.

376. ———. General intelligence objectively determined and measured, ibid., pp. 201–92.

377. ———. The theory of two factors, Psychol. Rev., **21**:101–15, 1914.

378. ———. The abilities of man. New York: Macmillan Co., 1927. Pp. vi+448.

379. ———. The factor theory and its troubles, J. Ed. Psychol., **25**:383–91, 1934.

380. ———. Thurstone's work re-worked, ibid., **30**:1–16, 1939.

381. Spence, K. W. The role of secondary reinforcement in delayed reward learning, Psychol. Rev., **54**:1–8, 1947.

382. Stellar, E.; Morgan, C. T.; and Yarosh, M. Cortical localization of symbolic processes in the rat, J. Comp. Psychol., **34**:107–26, 1942.

383. Stern, W. The psychological methods of testing intelligence. Baltimore: Warwick & York, 1914. Pp. vii+160.

384. Stern, W., and Wiegmann, O. Methodensammlung zur Intelligenzprüfung von Kindern und Jugendlichen, Ztschr. f. ang. Psychol., No. 20, 1920. Pp. v+256.

385. Stickney, J. C., and Van Liere, E. J. Acclimatization to low oxygen tensions in relation to gastric emptying, Am. J. Physiol., **137**:160–63, 1942.

386. Stoddard, G. The meaning of intelligence. New York: Macmillan Co., 1943. Pp. ix+504.

387. Strauss, A. A., and Werner, H. Disorders of conceptual thinking in the brain-injured child, J. Nerv. & Ment. Dis., 96:153–72, 1942.

388. ———. Comparative psychopathology of the brain-injured child and the traumatic brain-injured adult, Am. J. Psychiat., 99:835–38, 1943.

389. Strauss, H. Uber die hirnlokalisatorische Bedeutung des einseitigen Ausfalls des optokinetischen Nystagmus und der hemianopischen Aufmerksamkeitsschwäche, Ztschr. f. d. ges. Neurol. u. Psychiat., 143:427–35, 1933.

390. Strauss, I., and Savitsky, N. Head injury—neurologic and psychiatric aspects, Arch. Neurol. & Psychiat., 31:893–955, 1934.

391 Ström-Olsen, R.; Last, S. L.; Brody, M. B.; and Knight, G. C. Prefrontal leukotomy: results in 30 cases of mental disorder, with observations on surgical technic, J. Ment. Sc., 89:165–81, 1943.

392. Strong, O. S. Unsolved problems suggested by cerebellar connections and cerebellar histology, Arch. Neurol. & Psychiat., 19:1–30, 1928.

393. Terman, L. M. The measurement of intelligence: an explanation of and a complete guide for the use of the Stanford revision and extension of the Binet-Simon intelligence scale. Boston: Houghton Mifflin Co., 1916. Pp. xviii+362.

394. Thompson, J. The ability of children of different grade levels to generalize on sorting tests, J. Psychol., 11:119–26, 1941.

395. Thomson, G. H. The factorial analysis of human ability. Boston: Houghton Mifflin Co., 1939. Pp. xv+326.

396. Thorndike, E. L. Intelligence and its measurement: a symposium, I, J. Ed. Psychol., 12:124–27, 1921.

397. Thorndike, E. L.; Bregman, E. O.; Cobb, M. V.; *et al.* The measurement of intelligence. New York: Teachers College, Columbia University, 1927. Pp. xxvi+616.

398. Thurstone, L. L. Multiple factor analysis, Psychol. Rev., 38:406–27, 1931.

399. ———. Unitary abilities, J. Gen. Psychol., 11:126–32, 1934.

400. ———. The vectors of mind. Chicago: University of Chicago Press, 1935. Pp. xi+266.

401. ———. Primary mental abilities, Psychometric Monog. No. 1. Chicago: University of Chicago Press, 1938. Pp. ix+121.

402. ———. Second-order factors, Psychometrika, 9:71–100, 1944.

403. ———. Multiple-factor analysis. Chicago: University of Chicago Press, 1947. Pp. xix+535.

404. Tolman, E. C. Purposive behavior in animals and men. New York: Century Co., 1932. Pp. xiv+463.

405. Toman, J. Flicker potentials and the alpha rhythm in man, J. Neurophysiol., 4:51–61, 1941.

406. Tredgold, A. F. Mental deficiency. Baltimore: William Wood & Co., 1922. Pp. xx+564.

407. Troland, L. T. The mystery of mind. New York: D. Van Nostrand Co., 1926. Pp. xi+253.

408. Tryon, R. C. Studies in individual differences in maze ability. I. Measurement of the reliability of individual differences, J. Comp. Psychol., 11:145–70, 1930.

409. ———. Studies in individual differences in maze ability. IV. The constancy of individual differences: correlation between learning and relearning, ibid., 12:303–45, 1931.

410. Visser, J. A., and Rademaker, G. G. J. Die optischen Reaktionen grosshirnloser Tauben, Arch. néerl. de physiol., 19:482–501, 1934.

411. WAGENEN, W. P. VAN, and HERREN, R. Y. Surgical division of commissural pathways in the corpus callosum, Arch. Neurol. & Psychiat., **44**:740–59, 1940.
412. WALKER, A. E. The primate thalamus. Chicago: University of Chicago Press, 1938. Pp. xxiii+321.
413. ———. A cytoarchitectural study of the prefrontal area of the macaque monkey, J. Comp. Neurol., **73**:59–86, 1940.
414. ———. The medial thalamic nucleus: a comparative anatomical, physiological and clinical study of the nucleus medialis dorsalis thalami, ibid., pp. 87–115.
415. WALKER, A. E., and HALSTEAD, W. C. Neurological and psychological effects of cerebral injuries. In: Medicine and the war, pp. 132–49. Chicago: University of Chicago Press, 1944.
416. WALKER, A. E.; KOLLROS, J. J.; and CASE, T. J. The physiological basis of concussion, J. Neurosurg., **1**:103–15, 1944.
417. WALKER, A. E.; WOOLF, J. I.; HALSTEAD, W. C.; and CASE, T. J. Mechanism of temporal fusion effect of photic stimulation on electrical activity of visual structures, J. Neurophysiol., **6**:213–20, 1943.
418. ———. Photic driving, Arch. Neurol. & Psychiat., **52**:117–25, 1944.
419. WALLS, G. L. The vertebrate eye. Bloomfield Hills, Mich.: Cranbrook Institute of Science, 1942. Pp. xiv+785.
420. ———. The visual cells and their history. In: KLÜVER, H. (ed.), Biol. Symposia, **7**:203–51, 1942.
421. WANG, G. H. Action potentials of the visual cortex and the superior colliculus induced by stimulation of the retina with light, Chinese J. Physiol., **8**:121–44, 1934.
422. WATTS, J. W., and FREEMAN, W. Psychosurgery, J. Nerv. & Ment. Dis., **88**:589–601, 1938.
423. ———. Intelligence following prefrontal lobotomy in obsessive tension states, J. Neurosurg., **1**:291–96, 1944.
424. WECHSLER, D. The measurement of adult intelligence. Baltimore: Williams & Wilkins, 1939. Pp. ix+229.
425. WEIGL, E. Zur Psychologie sogenannter Abstraktionsprozesse, Ztschr. f. Psychol., **103**:1–45, 257–322, 1927.
426. WERNER, H. Perception of spatial relationship in mentally deficient children, J. Genet. Psychol., **57**:93–100, 1940.
427. WERNER, H., and THUMA, B. D. Critical flicker-frequency in children with brain injury, Am. J. Psychol., **55**:394–99, 1942.
428. WERTHEIMER, M. Experimentelle Studien über das Sehen von Bewegung, Ztschr. f. Psychol., **61**:161–265, 1912.
429. WHEATLEY, M. D. The hypothalamus and affective behavior in cats: a study of the effect of experimental lesions, with anatomical correlations, Arch. Neurol. & Psychiat., **52**:298–316, 1944.
430. WHITE, W. A. Outlines of psychiatry. Washington: Nerv. & Ment. Dis. Pub. Co., 1924. Pp. vii+388.
431. WILDE, J. F. Narco-analysis in the treatment of war neuroses, Brit. M. J., **2**:4–13, 1942.
432. WILLIAMS, A. C., JR. Facilitation of the alpha rhythm of the electroencephalogram, J. Exper. Psychol., **26**:413–22, 1940.
433. WOOLF, J. I.; WALKER, A. E.; KNOX, G. W.; and HALSTEAD, W. C. The effect of lesions of the visual system on photic driving, J. Neuropath. & Exper. Neurol., **4**:59–67, 1945.

434. YACORZYNSKI, G. K. An evaluation of the postulates underlying the Babcock deterioration test, Psychol. Rev., **48**:261–67, 1941.
435. ———. Differential modification of post-rotational nystagmus of pigeons with cerebral lesions, Comp. Psychol. Monog., **19**, No. 2, 38, 1946.
436. YACORZYNSKI, G. K., and DAVIS, L. An experimental study of the functions of the frontal lobes in man, Psychosom. Med., **7**:97–107, 1945.
437. YACORZYNSKI, G. K.; HALSTEAD, W. C.; and FEARING, F. A quantitative method for the measurement of equilibration of pigeons with cerebral, cerebellar, or labyrinthine lesions, J. Gen. Psychol., **25**:75–83, 1941.
438. ———. Relationship between the experimental reduction of vestibular nystagmus and equilibrium, J. Psychol., **11**:161–71, 1941.
439. ZIEGLER, L. H. Bilateral prefrontal lobotomy, Rev. mex. psiquiat., neurol. y med. leg., **10**:3–9, 1943. Also: Am. J. Psychiat., **100**:178–79, 1943.

INDEXES

AUTHOR INDEX

SUBJECT INDEX